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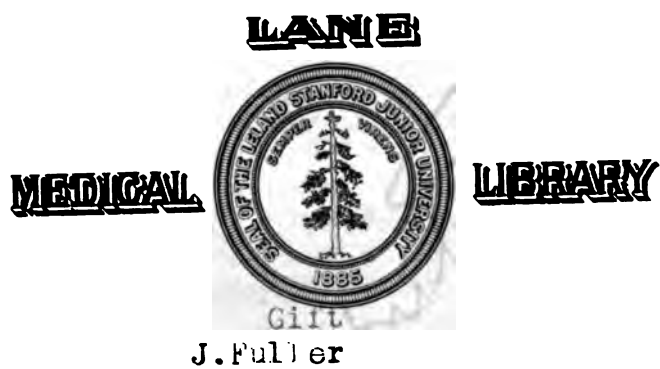
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THE DISEASES OF INFANCY  
AND CHILDHOOD



# THE DISEASES OF INFANCY AND CHILDHOOD

FOR THE USE OF STUDENTS  
AND PRACTITIONERS OF MEDICINE

BY

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THE GESELLSCHAFT FÜR INNERE MEDIZIN UND KINDERHEILKUNDE,  
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SIXTH EDITION, FULLY REVISED

*WITH TWO HUNDRED AND FORTY ILLUSTRATIONS  
INCLUDING EIGHT COLOURED PLATES*

NEW YORK AND LONDON  
D. APPLETON AND COMPANY

1911

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PRINTED AT THE APPLETON PRESS  
NEW YORK, U. S. A.

Y51A9B1 1911

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1911

TO  
VIRGIL P. GIBNEY, M.D., LL.D.,  
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SURGEONS (COLUMBIA UNIVERSITY), NEW YORK; SURGEON-IN-CHIEF  
TO THE HOSPITAL FOR THE RUPTURED AND CRIPPLED,  
THIS VOLUME IS INSCRIBED  
AS A TRIBUTE TO HIS PERSONAL WORTH AND HIGH PROFESSIONAL ATTAINMENTS,  
AND IN GRATEFUL REMEMBRANCE OF MANY ACTS OF KINDNESS  
BY THE AUTHOR.

29625





## PREFACE TO THE SIXTH EDITION.

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IN the preparation of this edition the author has associated with him Dr. John Howland, his former assistant, who will hereafter be connected with the work as joint author.

Progress along many lines in pædiatrics has been rapid during the last two years. To make room for new knowledge without unduly enlarging the size of the volume has made it necessary to cut out about seventy-five pages of old material. It is believed that this has been accomplished without impairing the value of the chapters which have been abridged. The decision of the publishers to make entirely new plates has made this comparatively easy.

There is scarcely a page in the book which has not been subject to some revision. Many articles have been entirely rewritten and several new ones appear for the first time in this edition. The greater part of the new material will be found in the chapters upon Nutrition and Infant Feeding, Infant Mortality, Intestinal Intoxication, Pyloric Stenosis, Appendicitis, Acute Peritonitis, Endocarditis and Pericarditis, Cerebro-spinal and Other Forms of Acute Meningitis, Acute Poliomyelitis, Hereditary Syphilis and Tuberculosis.

A number of the old illustrations have been omitted as no longer necessary; others have been replaced by better ones. In all, thirty-six new illustrations have been introduced, including twelve radiographs. All illustrations are from original sources unless otherwise stated.

The authors desire to acknowledge their indebtedness to Dr. F. H. Bartlett for much assistance rendered in every way in the work of revision; to Dr. H. H. Mason for correction of the proof sheets, and to Dr. N. C. Holt for the preparation of the index.

14 WEST FIFTY-FIFTH STREET,  
NEW YORK.



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# THE DISEASES OF INFANCY AND CHILDHOOD.

## PART I.

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### CHAPTER I.

#### *HYGIENE AND GENERAL CARE OF INFANTS AND YOUNG CHILDREN.*

THE physical development of the child is essentially the product of the three factors—inheritance, surroundings, and food. The first of these it is beyond the physician's power to alter; the second is largely and the third almost entirely within his control, at least in the more intelligent classes of society. These two subjects, infant hygiene and infant feeding, are the most important departments of pædiatrics.

**The Care of the Newly-Born Child.**—After the ligature of the cord the child should be wrapped in a thick blanket and placed in a warm room. In hospital practice the eyes should be cleansed with absorbent cotton and water which has been boiled, and then two or three drops of a two-per-cent solution of nitrate of silver, after Credé's method, instilled into each eye by means of a glass rod or eye-dropper. In private practice a ten-per-cent solution of argyrol may be substituted, unless the mother has had a purulent vaginal discharge, in which case the silver solution should always be used. The bath should now be given in a warm room; the body being first oiled thoroughly in order to remove the vernix caseosa and then washed in water at a temperature of 100° F. The mouth should be cleansed with sterile water and a soft cloth, and no violence employed. The cord may be covered with sterilised talcum or bismuth powder, and wrapped in sterile gauze or surgeon's lint. The abdomen should now be enveloped in a flannel band, eight or ten inches wide, and pinned rather snugly. Before dressing is completed, the child should be submitted to a thorough examination for injuries received during delivery, congenital deformities, also as to the condition of the respiration, circulation, etc.

After dressing, the child should be placed in his crib and covered with blankets, and if the feet are cold, or the fingers and lips a little blue, he should be surrounded by hot-water bottles covered with flannels, and placed near, but not in contact with, the body. The crib should be placed



in a quiet, darkened room. The young infant should not occupy the same bed as the mother, unless he greatly needs the warmth of her body, other means of artificial heat not being at hand.

The cord should be kept dry and disturbed as little as possible until it falls off. Under ordinary circumstances the cord separates from the fourth to the seventh day, the average being the fifth day. The stump should then be covered with the sterilised talcum or bismuth powder, and a pad of sterile gauze about one-fourth of an inch thick and two inches square applied and secured in position by means of the abdominal band. The purpose of this is to prevent umbilical hernia. The pad should be continued for the first month. The use of stronger antiseptic dressings than those recommended is somewhat objectionable, since it preserves the cord too long and delays separation. The full bath should not be given until the cord has separated.

The physician should always see to it that the infant cries enough to keep the lungs properly expanded.

The question of food for the newly-born infant is considered in the chapter upon infant feeding.

**Bathing.**—For the first few months the bath should be given at 98° F. The room should be warm, preferably there should be an open fire. The bath should be short and the body dried quickly, without too vigorous rubbing. The addition of salt to the bath is an advantage where the skin is unusually delicate or excoriations are present. One large handful should be used to a gallon of water. By the sixth month the temperature of the bath for healthy infants may be lowered to 95° F., and by the end of the first year to 90° F. Older children who are healthy should be sponged or douched for a moment at the close of the tepid bath with water at 65° or 70° F. During childhood the warm bath is preferably given at night. In the morning a cold sponge bath is desirable. This should be given in a warm room and while the child stands in a tub partly filled with warm water. This cold sponge should last but half a minute, and be followed by a brisk rubbing of the entire body.

In some young infants and even older children there is no proper reaction after the bath, even when given at the temperatures mentioned; children being pale, slightly blue about the lips and under the eyes. All tub bathing, and especially all cold bathing, should then be stopped, since a continuance can only be a drain upon the child's vitality.

**Clothing.**—The clothing of infants should be light, warm, non-irritating to the skin, and loose enough to allow free motion of the extremities; nor should bands be pinned so tightly about the trunk as to embarrass the movements either of the chest or of the abdomen. The chest should be covered with a woollen shirt, high in the neck and with long sleeves. All petticoats should be supported from the shoulders and not from waistbands. Canton flannel and stockinet are both superior as

absorbents to the more commonly used linen diapers. Stockinet has the advantage of being very soft and pliable. Care should be taken that in infants the feet be kept warm. If the circulation is very poor, a bag of hot water should always be in the crib. Cold feet are responsible for many attacks of colic.

The abdominal band is usually worn during infancy. It cannot be considered in any sense a necessity after the first few months, excepting in cases of very thin infants whose supply of fat in the abdominal walls is an insufficient protection to the viscera. For the first few weeks a band of plain flannel is to be preferred; later, a knitted band with shoulder-straps. The fashion of low neck and short sleeves for infants and very young children has fortunately passed away—let us hope, never to return.

During the summer the outer clothing should be light and the under clothing of the thinnest flannel or gauze. The changes in the temperature of morning and evening may be met by extra wraps. The custom of allowing young children to go with legs bare has many enthusiastic advocates; while it may not be objectionable during the heat of summer, its advantages at any season are very questionable in a changeable climate like that of New York or the Atlantic coast. Many delicate children are certainly injured by such ill-advised attempts at hardening.

The night clothing of infants should be similar to that worn during the day, but should be loose, the material being of the lightest flannel. The night clothing for older children should consist of a thin woollen shirt and a union suit with waist and trousers, and in some cases with feet, if there is a tendency to get outside the coverings. The common mistake is to overload all children, but especially infants, with covering at night. This is an explanation of much of the restless sleep which is seen particularly in delicate children.

**Care of the Eyes.**—During the first few days at the daily bath the eyes should be cleansed with a saturated solution of boric acid. They should be carefully protected from too strong light during early infancy. It is desirable that a child should always sleep in a darkened room.

**Care of the Mouth and Teeth.**—The mouth of the newly-born infant should be gently cleansed at each morning bath with boiled water and a soft cloth. On the first appearance of thrush the mouth should be washed after every feeding with a solution of bicarbonate of soda or boric acid (ten grains to the ounce). It should be applied with a swab made by twisting a bit of cotton upon a wooden toothpick, and not by the nurse's finger. Harm is often done by the use of too much zeal in cleansing the mouth of a young infant.

The primary teeth as well as those of the permanent set should receive daily attention. Too often they are neglected altogether. Dirty teeth are likely sooner or later to become carious; and carious teeth, besides

being a cause of bad breath and neuralgia, are a constant menace to the health of the child, since they may harbour infectious germs of all varieties. Such teeth should either be filled or removed.

**Care of the Skin.**—The skin of a young infant is exceedingly delicate, and excoriations, intertrigo, and eczema are of very common occurrence. These conditions are much easier of prevention than of cure. The first essential in the care of the skin is cleanliness, and this must be secured without the use of strong soaps or too much rubbing. Napkins must be removed as soon as soiled or wet. Some bland absorbent powder, like starch, talcum, or the stearate of zinc, should be used in all the folds of the skin, in the neck, in the axillæ, groins, and about the genitals, and in the folds of the thighs, particularly in very fat infants. If plain water produces an undue amount of irritation, the salt or bran bath should be employed.

**Care of the Genital Organs.**—The female genitals need but little attention in young children, excepting as to cleanliness. This is more often neglected in older children than in infants.

In males the prepuce should receive attention during the first few weeks of life. If the foreskin is long and the preputial orifice small, circumcision should be done. If it is not long, but is only adherent, these adhesions should be broken up, the parts thoroughly cleansed and the foreskin retracted daily until there is no disposition to a recurrence of the adhesions. These operations will be discussed more at length in a subsequent chapter. The only thing to be emphasised in the present connection is that the prepuce should receive proper attention in early infancy, since this can now be done with less pain and discomfort to the child, and at the same time better results are obtained. If this matter is neglected during infancy, it is apt to be overlooked until harm has been produced by local or reflex irritation which phimosis or adherent prepuce may have excited.

**Vaccination.**—This, although considered elsewhere, should be mentioned in this connection as among the things requiring the physician's attention during the first months of life.

**Training to Proper Control of Rectum and Bladder.**—It is surprising to see what can be accomplished by intelligent efforts at training in these particulars. An infant can often be trained at three months to have its movements from the bowels when placed upon a small chamber. This not only saves a great amount of washing of napkins, but there is soon formed a habit of having the bowels move at a regular time or times each day. The infant must be put upon the chamber soon after his feeding. The importance of training young children to regular habits regarding evacuations from the bowels can hardly be overestimated. It should be impressed upon every mother and nurse by the physician, and especially the necessity of beginning training during infancy. Much of

course will depend upon the food and the digestion; but habit is a very large factor in the case.

The training of the bladder is not quite so important, but the proper education of this organ adds much to the comfort of the child and the ease with which it is cared for. Before the end of the first year many intelligent children can be trained to indicate a desire to empty the bladder. Many mothers and nurses succeed in training children so well that by the tenth or eleventh month napkins are dispensed with during the day. On the other hand, it is very common to see children of two and even two and a half years still wearing napkins because of the lack of proper training. Before it has reached the age of three years a healthy child will usually go from 10 P.M. until morning without emptying the bladder. The annoyance and discomfort from the neglect of early training in this particular are very great. Night feeding is responsible for much of the difficulty experienced in training children to hold the water during the night.

**General Hygiene of the Nervous System.**—Great injury is done to the nervous system of children by the influences with which they are surrounded during infancy, especially during the first year. The brain grows more during the first two years than in all the rest of life. Normal healthy development of the nervous centres demands quiet, rest, peaceful surroundings, and freedom from everything which causes excitement or undue stimulation.

The steadily increasing frequency of functional nervous diseases among young children is one of the most powerful arguments for greater attention by physicians to the subject of the hygiene of the nervous system during infancy. Most parents err through ignorance. Playing with young children, stimulating to laughter and exciting them by sights, sounds, or movements until they shriek with apparent delight, may be a source of amusement to fond parents and admiring spectators, but it is almost invariably an injury to the child. This is especially harmful when done in the evening. It is the plain duty of the physician to enlighten parents upon this point, and insist that the infant shall be kept quiet, and that all such playing and romping as has been referred to shall, during the first year at least, be absolutely prohibited.

**Sleep.**—The sleep of the newly-born infant is profound for the first two or three days and under normal conditions almost continuous. In the case of prolonged or tedious labour, or where from any cause undue compression has been exerted upon the head, it may approach the condition of semi-coma for twenty-four or forty-eight hours. This may be so deep as to excite apprehensions of serious brain lesions. If, however, there are associated with it no convulsions and no rigidity, this early stupor usually passes away on the second or third day.

The sleep of early infancy is quiet and peaceful, but not very deep

after the first month. After the third year the heavy sleep of childhood is commonly seen. A healthy infant during the first few weeks sleeps from twenty to twenty-two hours out of the twenty-four, waking only from hunger, discomfort, or pain. During the first six months a healthy infant will usually sleep from sixteen to eighteen hours a day, the waking periods being only from half an hour to two hours long. At the age of one year most infants sleep from fourteen to fifteen hours, viz., from eleven to twelve hours at night, and two or three hours during the day, usually in two naps. When two years old usually thirteen to fourteen hours' sleep is taken; eleven or twelve hours at night and one or two hours during the day, generally in a single nap. At the age of four years children require from eleven to twelve hours' sleep. It is always desirable, and in most cases with regularity it is possible, to keep up the daily nap until children are four years old. From six to ten years the amount of sleep required is ten or eleven hours, and from ten to sixteen years nine hours should be the minimum.

Training in proper habits of sleep should be begun at birth. From the outset an infant should be accustomed to being put into his crib while awake and to go to sleep of his own accord. Rocking and all other habits of this sort are useless and may even be harmful. An infant should not be allowed to sleep on the breast of the nurse, nor with the nipple of the bottle in his mouth. Other devices for putting infants to sleep, such as allowing the child to suck a rubber nipple or anything else, are positively injurious. If such means of inducing sleep are resorted to the infant soon acquires the habit of not sleeping without them. I have known of one instance where the habit of rocking during sleep was continued until the child was two years old; the moment the rocking was stopped the infant would wake, and in consequence this practice was continued by the devoted but misguided parents. A quiet, darkened room, a warm and comfortable bed, an appetite satisfied, and dry napkins are all that are needed to induce sleep in a healthy child.

The periods of sleep in young infants are usually from two to three hours long, with the exception of once or twice in the twenty-four hours, when a long sleep of five or six hours occurs. The purpose of training is to have the child take this long sleep at night. The habit of regular sleep is best established by wakening the infant regularly every two or two and a half hours during the day for feeding, and allowing it to sleep as long as possible during the night. This training goes hand-in-hand with regular habits of feeding. Such habits are easily formed if the plan be systematically followed from the outset.

By the fifth month all feeding between 10 P.M. and 7 A.M. should be discontinued. If this is done most infants can be trained by this time to sleep all night. If the room is lighted, and the child taken from the crib or rocked or fed as soon as he awakens at night, there is no such thing

as the formation of good habits of sleep. Regularity in sleep and feeding not only make the care of young infants very much easier, but they are of a good deal of importance for the health of the child.

The causes of disturbed or irregular sleep in young infants are mainly two—hunger and indigestion. In nursing infants it is usually the former; in those artificially fed usually the latter. Sleeplessness from hunger is often seen in children who are nursed thirty or forty minutes and then fall asleep, but wake in fifteen or twenty minutes crying and fretful. After being quieted they may fall asleep again for half an hour, but wake at short intervals. The peaceful sleep of two or three hours which should follow a proper feeding is never seen. With this restlessness, in indigestion other signs are usually present, stationary weight, etc. The disturbed sleep due to overfeeding shows itself by much the same symptoms, excepting that the first sleep after the meal is usually longer.

**Exercise.**—This is no less important in infancy than in later childhood. An infant gets his exercise in the lusty cry which follows the cool sponge of the bath, in kicking his legs about, waving his arms, etc. By these means pulmonary expansion and muscular development are increased and the general nutrition promoted. An infant's clothing should be such as not to interfere with his exercise. Confinement of the legs should not be permitted. In hospital practice I have often had a chance to observe the bad results which follow when very young infants are allowed to lie in the cribs nearly all the time. Little by little the vital processes flag, the cry becomes feeble, the weight is first stationary, then there is a steady loss. The appetite fails so that food is at first taken without relish, then at times altogether refused; later, vomiting ensues and other symptoms of indigestion. This, in many cases, is the beginning of a steady downward course which goes on until a condition of hopeless marasmus is reached. Such infants must be taken up every few hours and carried about the wards; the position should be frequently changed, and general friction of the entire body employed at least twice a day. Every means must be made use of to stimulate the vital activity. The value of systematic attention to these matters cannot be overestimated in hospitals for infants. Infants who are old enough to creep or stand usually take sufficient exercise unless they are restrained. At this age they should be allowed to do what they are eager to do. Every facility should be afforded for using their muscles. Exercise may be encouraged by placing upon the floor in a warm room a mattress or a thick "comfortable," and allowing the infant to roll and tumble upon it at will. A large bed may answer the same purpose.

In older children every form of out-of-door exercise should be encouraged—ball, tennis, and all running games, horseback riding, the bicycle, tricycle, swimming, coasting, and skating. Up to the eleventh year no difference need be made in the exercise of the two sexes. Companion-

ship is a necessity. Children brought up alone are at a great disadvantage in this respect, and are not likely to get as much exercise as they require. The amount of exercise allowed delicate children should be regulated with some degree of care. It may be carried to the point of moderate muscular fatigue, but never to muscular exhaustion. The latter is particularly likely to be the case in competitive games.

Exercise should have reference to the symmetrical development of the whole body. In prescribing it the specific needs of the individual child should be considered. By carefully regulated exercises very much may be done to check such deformities as round shoulders and slight lateral curvature of the spine, and also to develop narrow chests and feeble thoracic muscles. For purposes like these, gymnastics are exceedingly valuable to supplement out-of-door exercise, but they can never take their place.

There are two important points with reference to exercise indoors. First, the playroom should be cool—about 60° F. Secondly, during all active exercise the clothing should be loose and light, so as to allow the freest possible motion of the body.

**Airing.**—In summer there can be no possible objection to a young infant being allowed out of doors at the end of the first week. He should be kept in the open air as much as possible during the day. In the fall and spring this should not be permitted until the child is at least a month old, and then only when the out-of-door temperature is above 60° F. During his outing the head should be protected from the wind and the eyes from the sun. The duration of the outing at first should be only fifteen or twenty minutes, the time being gradually lengthened to two or three hours. The child should be gradually accustomed to change of temperature in the room by opening wide the windows for a few minutes each day even before it is taken out of doors, the child being dressed meanwhile as for an outing. In the case of children born late in the fall or in the winter this means of giving fresh air may be advantageously begun at one month and followed throughout the first winter. It is only necessary in all such cases that the changes be made very gradually both as to the length of the airing and as to the temperature. The great advantage of this plan over that more commonly followed of keeping young infants closely housed for the first six months in case they are born in the fall or early winter, I can positively affirm from quite a wide observation of both methods. It is a matter of very serious importance that every infant be furnished an abundance of pure fresh air in winter as well as in summer. When the plan above outlined is carefully and judiciously followed, the tendency to catarrhal affection instead of being increased is thereby greatly lessened.

When four or five months old, there is no reason why a healthy child should not go out of doors on pleasant days if the temperature is no

below 20° F. While there is a prejudice on the part of many mothers and some physicians against a child's sleeping out of doors in cold weather, it is a practice which I have always urged upon mothers, and have never seen followed by any but the most beneficial results. The days of all others when infants and very young children should not be out of doors are when there are high winds, especially those from the northeast, an atmosphere of melting snow, and during severe storms. Delicate infants must of course be more carefully guarded during the cold season. With most of these the plan of house-airing is all that should be attempted.

**Nursery.**—This should be the sunniest and best-ventilated room in the house. It is the physician's duty to see that proper attention is paid to the hygiene of the room in which the child spends at least four-fifths of his time during the first year, and two-thirds of his time during the first two or three years of life. Sunlight is absolutely indispensable. Sunny rooms always contain less organic matter and less humidity, and hence a room upon the north side of the house should always be avoided, preferably one in the second story should be chosen. Nothing which can in any way contaminate the air of the room should be allowed. There should be no drying of clothes or of napkins, and no plumbing. No food should be allowed to stand about the room. The gas should not be allowed to burn at night; a small wax night-light furnishes all that is needed in the nursery. If possible the heat should be from an open fire; the next best thing is the Franklin heater. Nothing in the room is worse than steam heat from a radiator unless it be a gas stove, which under no circumstances should be allowed, excepting possibly for a few minutes each morning during the bath.

The temperature of the room during the day should not be over 70° F. It is important that every nursery should have a thermometer, and that this and not the sensations of the nurse should be the guide. It is almost invariably true that the nursery is overheated. Often no other explanation can be found for chronic indigestion and falling weight excepting a nursery whose habitual temperature ranges from 75° to 80° F. At night for the first few weeks the temperature should not be allowed to fall below 65° F. After two months the night temperature may fall to 60° or even 50° F.

Free ventilation without draughts is an absolute necessity. This is best accomplished by ventilators in the windows, of which there are many excellent devices sold in the shops. While the child is absent from the room the windows should be widely opened and free airing of the nursery accomplished. The room should always be thoroughly aired at night before the child is put to bed. The window may be kept open after the third month. After the first year the window may be open, unless the outside temperature is as low as 20° F. If the window is



A more efficient means of furnishing artificial heat is by the electric pad. These small heaters are attached to an electric fixture like a drop light. A convenient size is ten by fifteen inches. The pad, which can be obtained of any electric supply company, is placed beneath two or three thicknesses of blanket, upon which the infant lies in its basket. Since the pads occasionally get out of order they must be used with some caution, as they have been known to burn the bed-clothes and even the baby.

With such means as those described it is possible to maintain the body temperature at normal even in a room kept at the ordinary temperature. It is preferable to have a warmer room; 75° or even 80° F is desirable for feeble infants. Adequate ventilation, however, is indispensable. With intelligent care excellent results can, however, often be obtained with no other means for maintaining heat than the padded basket and hot-water bottles; but the other accessories make the problem an easier one.

Premature infants should be fed without being removed from the basket, until they are strong enough to take the breast. The position should be frequently changed and some freedom of movement of the limbs permitted, but the infants should be handled as little as possible. The body should be oiled and fresh cotton applied every other day. The rectal temperature at first should be taken several times a day in order to be sure that sufficient artificial heat is being supplied, but not too much. The latter condition is one that often obtains. So long as the rectal temperature varies only between 98° and 100° F. one should be satisfied.

**Incubators.**—Personally, I have not found the usual small incubator a very satisfactory means of caring for the premature infant. The difficulties in successful operation are many and the dangers consequent upon the mode of ventilation are considerable. Except by persons experienced, their use is not to be advised. In hospitals with specially trained nurses they may give excellent results, but in the average home the simpler measures above described are much safer and quite efficient.

Every institution receiving and caring for premature infants should have a specially equipped room for that purpose. It should be of sufficient size to accommodate several patients. We have had such a room constructed in the Babies' Hospital which seems to fulfill all the requirements. The room has a floor space of thirteen by sixteen feet with ceiling eleven feet high. This is arranged for five infants, which gives each child 45 cubic feet of air. The cribs are separated by glass plates, which project three feet from the side wall and are four feet in height, forming for each infant a sort of alcove. The purpose of this is to diminish the chances of bed-to-bed infection. The room has double partition wall and double windows. The temperature is controlled by a thermostat.

regulator and is maintained at about 90° F. The room is provided with a special ventilating apparatus by means of which the entire air of the room can be changed in a few minutes. This is done several times a day. Such a room possesses all the advantages of the small incubator without any of its drawbacks. The infants are clothed in a single loose garment of absorbent cotton and cheese-cloth and lightly covered. In this room the normal body temperature is easily maintained. For wet-nursing, bathing, and changing of napkins, the children are removed to an anteroom which is kept at a temperature of about 75° F. When the bottle is given they are fed in their cribs. After reaching the weight of five and a half or six pounds they are removed to the anteroom for a few days, after which they are placed in the ward or sent home.

**Feeding.**—The feeding of the premature infant is not less important than the maintenance of heat and proper ventilation. Infants at eight months and those weighing five pounds or thereabouts can usually be made to take the breast after the first few days. Few below this age or weight will do so. Some will suck from a bottle, but the majority must be fed by other means. A medicine dropper may be used, or the Breck feeder<sup>1</sup> (Fig. 1); the smallest and feeblest, however, must be fed by gavage, using a funnel and small rubber catheter. The food should be slowly given; if rapidly, some is liable to be regurgitated, and this may produce attacks of asphyxia or even an aspiration pneumonia. The quantity of food and frequency of feeding will depend upon the size and age of the child. A seven months' baby weighing three and a half pounds should have, for the first twenty-four to thirty-six hours, only water, one to three teaspoonfuls every hour. Then regular food, half an ounce every hour, gradually increased to an ounce every two hours at the end of two weeks, and an ounce and a half every two hours at the end of three weeks.

Artificial feeding is seldom very successful with premature infants. With some of the larger and more vigorous, cow's milk modified according to the directions given in the chapters on Infant Feeding gives good results. I once succeeded with a child of three pounds two ounces. For most of them under four and a half pounds, breast-milk is essential. If the child is born near term, the mother may be able to nurse it. Occasionally this may be done at eight months, but seldom earlier, so that the milk of some other woman must usually be depended upon.



FIG. 1.—BRECK'S FEEDING-TUBE.

<sup>1</sup> Obtained at any of the Walker-Gordon laboratories.

As the premature baby requires only from six to twelve ounces of breast-milk a day for the first few weeks, this may be secured from some other nursing woman; a friend might be willing to furnish it or it could be purchased from any healthy woman who has an abundant supply. It is sufficient if it is drawn fresh twice a day, the utmost precautions, of course, being taken to secure cleanliness. At first equal parts of breast-milk and a four- or five-per-cent solution of milk sugar may be given, the degree of dilution being gradually lessened until pure milk is taken. Twelve feedings a day are usually necessary, the amount at one feeding may be from two drachms to one ounce depending upon the size, age and digestive powers of the infant. Since the breast-milk must always be diluted, at least at first, it is not important that the baby of the woman furnishing the milk should be of the same age as the foster infant. The milk of any woman whose baby is between one and eight months old will answer. I have successfully fed premature infants with breast-milk of women whose children were older than this. Another plan is to secure a wet-nurse and permit her to bring her own baby into the house. She pumps for the premature infant the required amount three or four times a day, and the rest of the time nurses her own child. In this way her flow of milk is maintained; but if the breasts are pumped exclusively the supply rapidly diminishes. The secretion of the milk of the mother may be promoted by her suckling the wet-nurse's baby or some other vigorous infant. The above are temporary expedients and in most instances need not be continued more than two or three weeks at the end of which time the mother may be able to nurse her own child.

The results with premature babies will depend very much upon how soon after birth they receive proper care. Immediately after birth measures should be taken to secure the best care and provide adequately for maintaining the body heat. If an incubator is to be used it should be in readiness, so that the child can be put into it as soon as it is breathing properly. The age and vigour of the infant are of the greatest importance in estimating the chances of survival. The following table gives Tarnier's statistics, showing the percentage of premature infants saved during a period of five years without the incubator, and during the succeeding five years with the incubator; also the percentage saved at the Sloane Hospital (New York), as published by Voorhees:

| AGE.                       | Tarnier saved<br>without incubators. | Tarnier saved<br>with incubators. | Voorhees<br>saved with<br>incubators. | Voorhees saved<br>excluding cases<br>dying a few<br>hours after birth |
|----------------------------|--------------------------------------|-----------------------------------|---------------------------------------|---|
| Born at 6 months . . . . . | 0.0%                                 | 16.0%                             | .....                                 | .....   |
| " " 6½ " . . . . .         | 21.5%                                | 36.6%                             | 22.0%                                 | 66.0%   |
| " " 7 " . . . . .          | 39.0%                                | 49.8%                             | 41.0%                                 | 71.0%   |
| " " 7½ " . . . . .         | 54.0%                                | 77.0%                             | 75.0%                                 | 89.0%   |
| " " 8 " . . . . .          | 78.0%                                | 88.8%                             | 70.0%                                 | 91.0%   |
| " " 8½ " . . . . .         | 88.0%                                | 96.0%                             | .....                                 | .....   |

Results will improve with the experience of the physician in the feeding and care of these very sensitive patients. Much is yet to be learned about them.

## CHAPTER II.

### *GROWTH AND DEVELOPMENT OF THE BODY.*

OBSERVATIONS upon growth and development are of the utmost importance during infancy and childhood. Only by this means are very many diseases detected in their incipiency. Early recognition carries with it in most cases the possibility of checking such pathological processes as, if allowed to go on, may affect the health not only in infancy but even throughout life.

By familiarity with what is normal, detection of the abnormal soon becomes easy. Investigation in regard to these subjects should be made a part of the physical examination of every child.

### WEIGHT.

The weight of the infant is the best means we have to measure his nutrition. It is as valuable a guide to the physician in infant feeding as is the temperature in a case of continued fever. Although the weight is not to be taken as the only guide to the child's condition, it is of such importance that we cannot afford to dispense with it during the first two years. It is a great advantage to keep up regular observations during childhood.

Weekly weighings should be made for the first six months, bi-weekly for the rest of the first year, and monthly during the second year. Delicate children should be weighed even more frequently. Balance scales only should be used. The spring scales are not reliable. A useful pattern of balance scales is shown in Fig. 2.

**Weight at Birth.**—The following figures are taken consecutively in nearly equal proportion from the records of the Nursery and Child's Hospital, the Sloane Maternity, and the New York Infant Asylum, and include only full-term children:



FIG. 2.

|   |                            |
|---|----------------------------|
| Average weight of 568 females . . . . . | 7.16 lbs. (3,260 grammes). |
| " " 590 males . . . . .                 | 7.55 " (3,400 " ).         |
| " " 1,158 infants . . . . .             | 7.35 " (3,330 " ).         |

**Weight Curve during the First Few Weeks.**—The accompanying chart represents the variations in weight for the first twenty days. The observations were made upon one hundred healthy, nursing infants, fifty males and fifty females, at the Nursery and Child's Hospital. The children were weighed daily during the period of observation. The

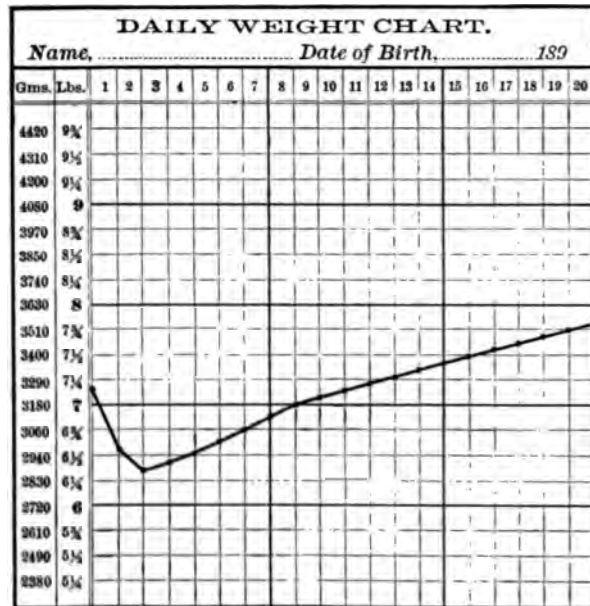


FIG. 3.—WEIGHT CURVE OF THE FIRST TWENTY DAYS.

average weight at birth was 7.1 pounds. The curve shows a very marked loss of weight on the first day and a slight loss on the second day, the lowest point being touched at the beginning of the third day, but from this time there was a steady gain. The average initial loss in these cases was ten ounces, being in each sex exactly eleven per cent of the body weight. In eight hundred and thirty-five cases, including the above mentioned, the average loss was nine and a half ounces. The loss of the first days is chiefly due to the discharge of the meconium in urine, but is in part from the excess of tissue waste over the nutriment derived from the breasts. After the third day, coincident with an abundant secretion of milk, there is a steady, daily increase in weight. If the milk is very scanty or is wanting altogether, the loss in weight continues.

The birth-weight of nursing children who thrive normally is regained on the average on the tenth day. The most frequent deviation from the normal curve consists in a continued loss or stationary weight after the third day. This may be due to acute illness, such as bronchitis, diarrhoea, pyæmia, or hæmorrhage, but in the majority of cases there is a disturbance of nutrition from improper or insufficient food.

The weight curve of infants who are artificially fed, even though they are strong and vigorous and the feeding properly done, rarely follows for the first month the same lines as that of nursing infants. We usually see an initial loss which is about the same as in nursing infants, then a period of nearly stationary weight lasting from one to two weeks.

Excessive loss in weight during the first few days from any cause whatsoever, seriously handicaps an infant during the first weeks of its life. The great importance of this has not been sufficiently appreciated.

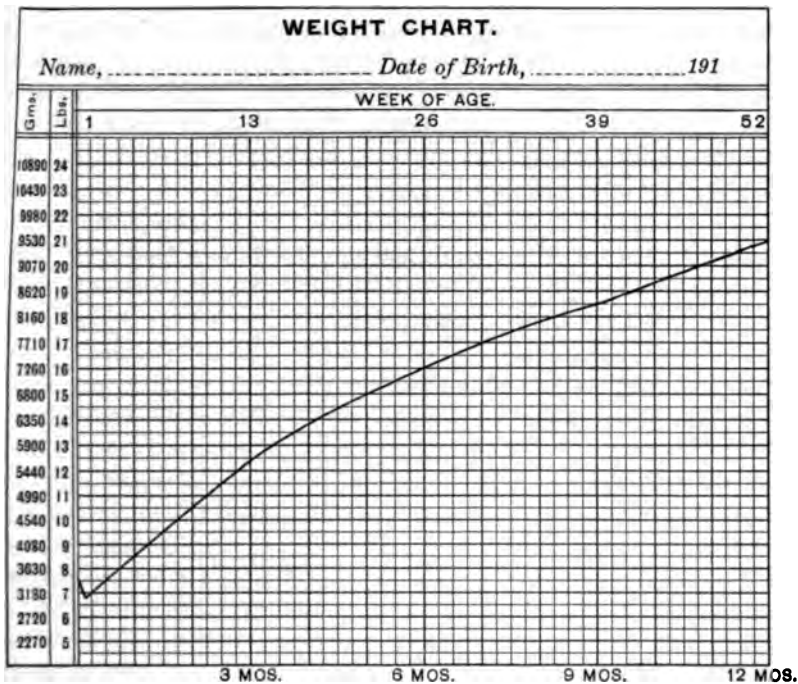


FIG. 4.—WEIGHT CURVE OF THE FIRST YEAR.<sup>1</sup>

**Weight Curve of the First Year.**—The curve of the accompanying chart is made up from complete weight charts of one hundred healthy nursing infants who were thriving and weighed every week, and the incomplete charts of about three hundred other infants. There are repre-

<sup>1</sup> Blank weight charts are made by Geo. L. Goodman & Co., 101 Beekman Street, New York.

sented in round numbers about twenty thousand observations on children under one year. The period of most rapid increase is during the first three months. It is slowest from the sixth to the ninth month. This curve is not to be regarded as a normal line, like the normal line of the temperature chart, but as an average line. An infant who is at birth a pound above the average may keep this distance above the line for the whole year; another, weighing one pound less than the average, may be as far below it. Girls throughout the year are on the average half a pound lighter than boys. No single child exactly follows the line all the way, but it is surprising how close to it a very large number of the cases come.

In artificially-fed infants—provided the feeding is properly done—the curve does not differ essentially from that of breast-fed infants, excepting in the slower gain of the first month, although this difference is usually made up before the sixth month is reached.

At the end of the first year the average child weighs nearly three times as much as at birth. Perfect health during the first year is seen only in children who are gaining steadily in weight. A child may not always gain rapidly, but it should gain steadily, and if it does not, something is wrong. All the conditions surrounding the infant should be investigated, but especially the food. One should not be satisfied unless the average weekly gain during the first six months is at least four ounces. In the second six months it may be slightly less. As a rule, a child who gains regularly in weight is thriving; an exception must, however, be made in the case of some infants who are fed chiefly upon carbohydrate foods.

**Weight from the Second to the Fifth Year.**—Comparatively few observations have been published upon the weight during this period. From nearly one thousand personal observations it appears that the normal gain of a healthy child is about six pounds during the second year, about five during the third year, and about four pounds during the fourth year; the actual weights are given in the large table on page 20. During this period the gain is rarely uniform after the first year. With most children it is slowest or the weight is stationary in the summer months, while the most rapid increase is usually seen in autumn. Throughout this period the girls gain in about the same ratio as boys, but remain on the average nearly one pound lighter. During almost every illness, no matter of what character, the gain in weight ceases, and usually there is a loss, the rapidity and extent of which are somewhat proportionate to the severity of the attack; but it is always much more rapid in diseases of the digestive tract than in any other form of illness.

**Weight of Older Children.**—The weights given in the table of children from five to fourteen years are from Bowditch. Observations were made upon children of American parentage in the public schools of Boston—

upon 4,327 boys and 3,681 girls.<sup>1</sup> It is to be remembered that these weights include the ordinary clothing, while those below five years are without clothing.<sup>2</sup>

The slowest gain is from the fifth to the eighth year, when it is about four pounds a year. From the eighth to the eleventh year it rises to about six pounds a year. Up to the eleventh year the two sexes gain in about the same ratio. From the eleventh to the thirteenth year the girls gain much more rapidly, passing the boys for the first time and maintaining this lead until the fifteenth year, when again the boys pass them.

## HEIGHT.

The figures showing the height at different ages are given in the following table. The measurements of infants at birth are taken in about equal numbers from the records of the New York Infant Asylum and the Sloane Maternity Hospital. They were made upon full-term infants.

|                                       |                          |
|---------------------------------------|--------------------------|
| Average length of 231 males . . . . . | 20.61 inches (52.5 cm.); |
| “ “ 211 females . . . . .             | 20.47 “ (52.2 “ );       |
| “ “ 442 infants . . . . .             | 20.54 “ (52.35 “ ).      |

The most rapid gain in length is in the first year. During this period the child grows on an average a little over eight inches (21 cm.). This gain is usually, but not always, proportionate to the increase in weight. During the second year the average increase is three and a half inches (9

<sup>1</sup> W. T. Porter has published (1894) observations made upon 14,744 children of American parentage in the public schools of St. Louis. His figures show quite a variation from those of Bowditch, and are as follows:

| AGE.              | BOYS' WEIGHT. |         | GIRLS' WEIGHT. |         |
|-------------------|---------------|---------|----------------|---------|
|                   | Kilos.        | Pounds. | Kilos.         | Pounds. |
| 6 years . . . . . | 19.66         | 43.2    | 18.76          | 41.3    |
| 7 “ . . . . .     | 21.67         | 47.7    | 20.82          | 45.8    |
| 8 “ . . . . .     | 23.91         | 52.6    | 22.71          | 50.0    |
| 9 “ . . . . .     | 26.08         | 57.4    | 25.07          | 55.1    |
| 10 “ . . . . .    | 28.49         | 62.7    | 27.43          | 60.3    |
| 11 “ . . . . .    | 31.26         | 68.8    | 29.93          | 65.8    |
| 12 “ . . . . .    | 33.45         | 73.6    | 33.17          | 73.0    |
| 13 “ . . . . .    | 35.96         | 79.1    | 38.29          | 84.2    |
| 14 “ . . . . .    | 40.34         | 88.7    | 43.12          | 94.9    |
| 15 “ . . . . .    | 47.25         | 103.9   | 46.90          | 103.2   |
| 16 “ . . . . .    | 52.10         | 114.6   | 50.06          | 110.1   |

<sup>2</sup> The average weight of the ordinary house clothing of school children, according to Bowditch, is at five years, 2.8 pounds for both sexes; at seven years, 3.5 for both sexes; at ten years, 5.7 pounds for boys and 4.5 pounds for girls; at thirteen years, 7.4 pounds for boys and 5.6 pounds for girls; at sixteen years, 9.7 pounds for boys and 8.1 pounds for girls. This must be deducted from weights given to obtain the net weight.



*Table showing Weight, Height, and Circumference of the Head and Chest from Birth to the Sixteenth Year.<sup>1</sup>*

| AGE.                       | Sex.   | WEIGHT.      |              | HEIGHT.     |              | CHEST.      |             | HEAD.       |             |
|----------------------------|--------|--------------|--------------|-------------|--------------|-------------|-------------|-------------|-------------|
|                            |        | Pounds.      | Kilos.       | Inches.     | Cm.          | Inches.     | Cm.         | Inches.     | Cm.         |
| Birth <sup>2</sup> .....   | Boys.  | <b>7.55</b>  | <b>3.43</b>  | <b>20.6</b> | <b>52.5</b>  | <b>13.4</b> | <b>34.2</b> | <b>13.9</b> | <b>35.5</b> |
|                            | Girls. | 7.16         | 3.26         | 20.5        | 52.2         | 13.0        | 33.2        | 13.5        | 34.5        |
| 6 months <sup>2</sup> ...  | Boys.  | <b>16.0</b>  | <b>7.26</b>  | <b>25.4</b> | <b>64.8</b>  | <b>16.5</b> | <b>42.0</b> | <b>17.0</b> | <b>43.5</b> |
|                            | Girls. | 15.5         | 7.03         | 25.0        | 63.6         | 16.1        | 41.0        | 16.6        | 42.2        |
| 12 months <sup>2</sup> ... | Boys.  | <b>21.0</b>  | <b>9.29</b>  | <b>29.0</b> | <b>73.8</b>  | <b>18.0</b> | <b>45.9</b> | <b>18.0</b> | <b>45.9</b> |
|                            | Girls. | 20.5         | 8.84         | 28.7        | 73.2         | 17.4        | 44.4        | 17.6        | 44.6        |
| 18 months <sup>2</sup> ... | Boys.  | <b>24.0</b>  | <b>10.35</b> | <b>30.0</b> | <b>76.3</b>  | <b>18.5</b> | <b>47.1</b> | <b>18.5</b> | <b>47.1</b> |
|                            | Girls. | 23.0         | 9.98         | 29.7        | 75.6         | 18.0        | 45.9        | 18.0        | 45.9        |
| 2 years <sup>2</sup> ..... | Boys.  | <b>27.0</b>  | <b>12.02</b> | <b>32.5</b> | <b>82.8</b>  | <b>19.0</b> | <b>48.4</b> | <b>18.9</b> | <b>48.2</b> |
|                            | Girls. | 26.0         | 11.56        | 32.5        | 82.8         | 18.5        | 47.0        | 18.6        | 47.2        |
| 3 years <sup>2</sup> ..... | Boys.  | <b>32.0</b>  | <b>14.14</b> | <b>35.0</b> | <b>89.1</b>  | <b>20.1</b> | <b>51.1</b> | <b>19.3</b> | <b>49.0</b> |
|                            | Girls. | 31.0         | 13.60        | 35.0        | 89.1         | 19.8        | 50.5        | 19.0        | 48.4        |
| 4 years <sup>2</sup> ..... | Boys.  | <b>36.0</b>  | <b>15.87</b> | <b>38.0</b> | <b>96.7</b>  | <b>20.7</b> | <b>52.8</b> | <b>19.7</b> | <b>50.3</b> |
|                            | Girls. | 35.0         | 15.41        | 38.0        | 96.7         | 20.7        | 52.2        | 19.5        | 49.6        |
| 5 years.....               | Boys.  | <b>41.2</b>  | <b>18.71</b> | <b>41.7</b> | <b>106.0</b> | <b>21.5</b> | <b>54.8</b> | <b>20.5</b> | <b>52.2</b> |
|                            | Girls. | 39.8         | 18.06        | 41.4        | 105.3        | 21.0        | 53.5        | 20.2        | 51.3        |
| 6 years.....               | Boys.  | <b>45.1</b>  | <b>20.48</b> | <b>44.1</b> | <b>112.0</b> | <b>23.2</b> | <b>59.1</b> | .....       | .....       |
|                            | Girls. | 43.8         | 19.87        | 43.6        | 110.9        | 22.8        | 58.3        | .....       | .....       |
| 7 years.....               | Boys.  | <b>49.5</b>  | <b>22.44</b> | <b>46.2</b> | <b>117.4</b> | <b>23.7</b> | <b>60.6</b> | .....       | .....       |
|                            | Girls. | 48.0         | 21.78        | 45.9        | 116.7        | 23.3        | 59.5        | .....       | .....       |
| 8 years.....               | Boys.  | <b>54.5</b>  | <b>24.70</b> | <b>48.2</b> | <b>122.3</b> | <b>24.4</b> | <b>62.2</b> | .....       | .....       |
|                            | Girls. | 52.9         | 24.01        | 48.0        | 122.1        | 23.8        | 60.8        | .....       | .....       |
| 9 years.....               | Boys.  | <b>60.0</b>  | <b>26.58</b> | <b>50.1</b> | <b>127.2</b> | <b>25.1</b> | <b>63.9</b> | .....       | .....       |
|                            | Girls. | 57.5         | 26.10        | 49.6        | 126.0        | 24.5        | 62.5        | .....       | .....       |
| 10 years.....              | Boys.  | <b>66.6</b>  | <b>30.22</b> | <b>52.2</b> | <b>132.6</b> | <b>25.8</b> | <b>65.6</b> | <b>21.0</b> | <b>53.5</b> |
|                            | Girls. | 64.1         | 29.07        | 51.8        | 131.5        | 24.7        | 63.0        | 20.7        | 52.8        |
| 11 years.....              | Boys.  | <b>72.4</b>  | <b>32.83</b> | <b>54.0</b> | <b>137.2</b> | <b>26.4</b> | <b>67.2</b> | .....       | .....       |
|                            | Girls. | 70.3         | 31.87        | 53.8        | 136.6        | 25.8        | 65.8        | .....       | .....       |
| 12 years.....              | Boys.  | <b>79.8</b>  | <b>36.21</b> | <b>55.8</b> | <b>141.7</b> | <b>27.0</b> | <b>68.8</b> | .....       | .....       |
|                            | Girls. | 81.4         | 36.90        | 57.1        | 145.2        | 26.8        | 68.3        | .....       | .....       |
| 13 years.....              | Boys.  | <b>88.3</b>  | <b>40.04</b> | <b>58.2</b> | <b>147.7</b> | <b>27.7</b> | <b>70.6</b> | .....       | .....       |
|                            | Girls. | 91.2         | 41.36        | 58.7        | 149.2        | 28.0        | 71.3        | .....       | .....       |
| 14 years.....              | Boys.  | <b>99.3</b>  | <b>45.03</b> | <b>61.0</b> | <b>155.1</b> | <b>28.8</b> | <b>73.3</b> | .....       | .....       |
|                            | Girls. | 100.3        | 45.50        | 60.3        | 153.2        | 29.2        | 74.1        | .....       | .....       |
| 15 years.....              | Boys.  | <b>110.8</b> | <b>50.26</b> | <b>63.0</b> | <b>159.9</b> | <b>30.0</b> | <b>76.6</b> | <b>21.8</b> | <b>55.5</b> |
|                            | Girls. | 108.4        | 49.17        | 61.4        | 155.9        | 30.3        | 76.8        | 21.5        | 54.8        |
| 16 years.....              | Boys.  | <b>123.7</b> | <b>56.09</b> | <b>65.6</b> | <b>166.5</b> | <b>31.2</b> | <b>79.2</b> | .....       | .....       |
|                            | Girls. | 113.0        | 51.24        | 61.7        | 156.7        | 30.8        | 78.8        | .....       | .....       |

<sup>1</sup> The observations of Boas (Science, April 12, 1895) upon 4,319 children over six years old show that first born exceed children born at a later period both in height and weight.

<sup>2</sup> These weights are without clothes; after five years clothes are included.

cm.). From this time on the rate of increase is quite uniform in both sexes until the eleventh year, it being between two and three inches a year.

After the eleventh year in girls and the twelfth in boys the growth is much more rapid. In height the girls exceed the boys at the twelfth and thirteenth years for the only time in their growth.

In the figures given in the preceding table those of five years and over are taken from Bowditch, the observations being made upon the same children as those whose weights were taken. The observations from six months to four years inclusive are from original sources, and are drawn from about five hundred cases. The height much more than the weight of children is modified by hereditary influences.

Rachitic children during infancy and early childhood are, as a rule, shorter than others. I have frequently measured such children during the third year who were six inches below the average for that age. The effect of malnutrition upon the length of the body is much less than upon the weight.

#### GROWTH OF THE EXTREMITIES AS COMPARED WITH THE TRUNK.

At birth the trunk is relatively long and the extremities short. The centre of the body at birth, according to one hundred observations made for me by Dr. Wilbur Ward at the Sloane Maternity Hospital, is three-quarters of an inch (2 cm.) below the centre of the umbilicus. Subsequently the growth of the extremities is much more rapid than that of the trunk. Thus I have found at birth the length of the lower extremities (measuring from the anterior superior spine of the ileum to the sole of the foot) to be forty-three per cent of the length of the body; at five years, fifty-four per cent, and at sixteen years, sixty per cent. The above figures are from one hundred and fifty observations, which, although not numerous enough for exact percentages, are still sufficient to give a very good idea of the general relation of the length of the extremities to that of the body as a whole. These facts are of some assistance in the diagnosis of diseases attended by abnormalities of growth, such as rickets, cretinism, and chondrodystrophy.

#### THE HEAD.

**Circumference.**—The average circumference of the head at birth in four hundred and forty-six full-term infants observed at the Sloane Maternity Hospital and New York Infant Asylum was as follows:

|  |                                      |
|--|--------------------------------------|
| Average circumference of the head, 231 males . . . . . | 13.90 inches (35.5 cm.);             |
| “ “ “ “ 215 females . . . . .                          | 13.52 “ (34.5 “ );                   |
| Total . . . . .  | 446 infants . . . 13.71 “ (35.0 “ ). |

The occipito-frontal measurement was the one taken.

The growth of the head is most rapid during the first year, the increase being about four inches (10 cm.). It is about half an inch a month during the early months, and a fourth of an inch a month during the later months of the first year. During the second year the increase is about one inch (2.5 cm.). From the second to the fifth year the growth is slower, being only about one and a half inches (4 cm.) for the three years. After the fifth year the increase in the circumference of the head is very slow (see table).

**Closure of the Sutures.**—The main sutures of the cranium are not commonly ossified before the end of the sixth month, and very frequently some mobility may be detected at the end of the ninth month. Distinct separation of the cranial bones after birth is abnormal. It is most frequently seen in premature and in syphilitic infants.

**Closure of the Fontanel.**—The posterior fontanel is usually obliterated by the end of the second month. The anterior fontanel under normal conditions closes on an average at about the eighteenth month. The usual variations are between the fourteenth and twenty-second months. At the end of the first year the fontanel is generally about one inch in diameter. An open fontanel at the end of the second year may be considered abnormal. The closure of the fontanel is not always early in well-nourished children, nor is it always delayed in those suffering from malnutrition. In very rare cases the anterior fontanel may either be closed at birth or may close during the first few weeks of life. Closure of the fontanel by the middle of the first year is often seen in cases of arrested cerebral development. This indicates a serious condition, usually microcephalus. Closure of the fontanel in the early months of the second year may be due to the slow growth of the brain in a child suffering from general malnutrition but otherwise normal.

By far the most frequent cause of delayed closure of the fontanel is rickets, in which condition it may be open up to the end of the third year. A large fontanel is one of the striking features of cretinism, and in untreated cases is often seen as late as the eighth year or later. In infancy an open fontanel with a rapid growth of the head should at once suggest hydrocephalus. There is an hereditary condition in which the fontanel remains open even to adult life. Two such cases in father and son were shown me by Marie in Paris. In both there was also lack of union between the two portions of the clavicle.

**Shape of the Head.**—The deformity which results from compression during labour usually disappears by the end of the first month. During the first year the head often becomes flattened at the occiput in consequence of the child's lying too much upon the back. This is easily remedied by changing his position. A slight obliquity of the head may result from a habitual position during nursing or sleep. A marked de-

gree of obliquity is sometimes congenital, but usually disappears by the third or fourth year.

The other abnormalities in the shape of the head are chiefly due to rickets and hydrocephalus, more rarely to congenital malformations of the brain. They will be considered in the chapter devoted to these topics.

Premature ossification of the sutures of the cranium occasionally gives rise to a very striking deformity of the head. I have seen two cases of



FIG. 5.—PREMATURE OSSIFICATION OF THE SAGITTAL SUTURE. Death at six weeks.

such deformity from premature ossification of the sagittal suture. The heads in both cases were very narrow and long in the antero-posterior diameter. The forehead was narrow, prominent, and slightly projecting. The illustration on this page shows the skull of one of these cases. There is a complete obliteration of the sagittal suture. In this case there was a wide separation of the sutures at the junction of the parietal and temporal bones.

#### THE CHEST.

The figures showing the circumference of the chest at the different periods of childhood have already been given. The measurements up to

and including five years are from personal observations, those from the sixth to the sixteenth are taken from Porter, and are drawn from observations on 31,371 school children. The measurement of the chest is that taken midway between full inspiration and expiration, and at the level of the nipples.

In the newly-born child the antero-posterior and the transverse diameters of the chest are nearly the same. As age advances, the transverse diameter increases very much more rapidly, so that the outline of the chest gradually assumes an elliptical shape, which it maintains during childhood.

At birth, the circumference of the chest is about one-half inch less than that of the head, but throughout infancy the two measurements are nearly the same. It is not until the third year that the average circumference of the chest exceeds that of the head. The chest measurement in infants is always much modified by the amount of fat; but, after making due allowance for this, a large chest always indicates a robust child and a small chest a delicate one. If at any age the circumference of the child's chest is found to be below the average, means should be taken, by gymnastics and otherwise, to develop it.

In infants deformities of the thorax result chiefly from rickets, sometimes from empyema, emphysema, and cardiac disease; in older children, from lateral curvature of the spine, or from Pott's disease. A peculiar deformity, usually congenital, but sometimes rachitic, is the funnel-shaped chest, the *Trichter-brust* of the Germans. It consists in a deep pit-like central depression at the lower end of the sternum. It is usually permanent.

#### THE ABDOMEN.

Throughout infancy the circumference of the abdomen is, as a rule, about the same as that of the chest. At the end of the second year the measurements of the head, chest, and abdomen are very often identical; after this time the chest measurement increases much more rapidly than the other two. Marked enlargement of the abdomen is seen in many varieties of chronic intestinal disorders. It is, however, most marked in the tympanites which so constantly accompanies rickets.

#### MUSCULAR DEVELOPMENT.

The first voluntary movements are usually in the fourth month, when the infant deliberately attempts to grasp some object placed before it. During the fourth month, as a rule, the head can be held erect when the trunk is supported. In many infants this is possible in the early part of the third month. At seven or eight months a healthy child is usually able to sit erect and support the trunk for several minutes.

In the ninth or tenth month are usually seen the first attempts to

bear the weight upon the feet. At eleven or twelve months a child usually stands with slight assistance. The first attempts at walking are commonly seen in the twelfth or thirteenth month. The average age at which children walk freely alone has been, in my experience, the fourteenth or fifteenth month. Quite wide variations are seen in healthy children. Very much depends upon the surroundings. I have known infants to walk at ten months and many others not until seventeen or eighteen months, although showing no evidences of disease, and although their development had not been retarded by previous illness. A very marked difference is seen in different families with respect to the time of walking.

The physician is often consulted because of backward muscular development, most frequently because the child is late in walking. General malnutrition, or any other severe or prolonged illness, may postpone for several months this or any of the other functions mentioned. When there is no such explanation of the backwardness, a child who does not hold up his head, sit alone, or make efforts to stand or walk at the proper time, should be submitted to a careful examination for mental deficiency or cerebral or spinal paralysis, but especially for rickets which is the most frequent explanation of the symptoms.

Contrivances for teaching infants to walk are unnecessary, and their effect may even be injurious. An infant should be allowed the greatest possible freedom in the use of his limbs. He should not be restrained from walking when inclined to do so, nor continually urged to walk when no voluntary attempts are made. Nothing short of mechanical restraint will prevent a healthy child from walking or standing when he is strong enough to do so.

#### DEVELOPMENT OF THE SPECIAL SENSES.<sup>1</sup>

**Sight.**—The newly-born infant avoids the light. The pupils contract in a light room, and if a bright light is brought before the eyes they close. During the first few weeks the infant indicates by every sign that excessive light is unpleasant. As early as the sixth day the eyes will sometimes follow a light in the room, and the child may even turn the head for this purpose. The muscles of the eyes of the newly-born infant act irregularly and not in harmony. Co-ordinate action for general purposes is not established until about the end of the third month. Even after this time inco-ordinate action is occasionally seen. The eyelids also move irregularly, and are often partly separated during sleep. The cornea is but slightly sensitive during the first weeks. In Preyer's child it was not until the third month that the lids closed when the water in

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<sup>1</sup> For many of the facts in this paragraph I am indebted to Preyer's *The Senses and the Will*, American edition, D. Appleton and Company.

the bath touched the lashes or the cornea. The recognition of objects seen is usually evident in the sixth month.

It is important that the room in which the newly-born child is placed should be darkened, and that for the first few weeks the eyes should be protected against strong light.

**Hearing.**—For the first twenty-four hours after birth infants are deaf. This deafness sometimes persists for several days. It is believed to be due to absence of air from the middle ear and to swelling of the mucous membrane which lines the tympanum. With the movements of respiration, air gradually finds its way into the middle ear, and the swelling subsides during the first few days. After this the hearing gradually improves, and during the early months of life it is very acute. The child starts at the slamming of a door, and even moderately loud noises will waken him from sleep. By the end of the second month he will sometimes turn his head in the direction from which the sound comes, and by the end of the third month this will usually be done. Demme found, in observations upon one hundred and fifty infants, that the voices of parents were recognised on an average at three and a half months.

Not only are the ears unusually sensitive to sound in infancy, but the impression produced upon the brain is often marked—very loud sounds causing great fright, and sometimes even, it is reported, convulsions.

**Touch.**—Tactile sensibility is present at birth, but is not highly developed except in the lips and tongue, where it is very acute for the obvious necessity, of sucking. After the third month it is fairly acute over the surface of the body generally. Two especially sensitive areas, according to Preyer, are the forehead and external auditory meatus.

Sensibility to painful impressions is present in early infancy, but very dull as compared with later childhood.

Temperature is also distinguished. This recognition is especially acute in the tongue. A young infant is often seen to refuse to take the bottle because the milk is only a few degrees too cold or too warm.

The localisation of sensory impressions comes later, probably not much before the middle of the sixth month, and is very imperfect throughout the first year.

**Taste.**—This is highly developed, even from birth. According to the experiments of Kussmaul, the ability to distinguish sweet, sour and bitter, exists in the newly-born child—sweet exciting sucking movements, and bitter, grimaces. A young infant detects with surprising accuracy the slightest variation in the taste of its food, and the smallest difference is often enough to cause it to refuse the bottle altogether. Sweet substances are always easily administered, and in combination with sirups even very bitter substances can be given; but to aromatic powders and elixirs he usually objects.

**Smell.**—Observations upon the sense of smell in newly-born infants are few and not altogether conclusive. Kroner's experiments appear to show that smell is present in the newly born. It has been noted to be especially acute in infants born blind. The sense of smell is developed much later than the other senses. Detection of fine differences in odours is not acquired until quite late in childhood.

### SPEECH.

There is a very wide variation in children with reference to the time of development of the function of speech. Girls, as a rule, talk from two to four months earlier than boys. Towards the end of the first year the average child begins with the words "papa," "mamma." By the end of the second year he is able to put words together in short sentences of two or three words. Progress in speech from this time is very rapid, each month showing great improvement. Names of persons are commonly first acquired, then the names of objects. Next to this the verbs are learned, and then adverbs and adjectives. Conjunctions, prepositions, and articles follow in order, and last of all the personal pronouns.

If a child of two years makes no attempt to speak, some mental defect may usually be inferred or that the child is a deaf mute.

### DENTITION.

The teeth are enclosed at birth in dental sacs which are situated in the gums. Superficially they are covered by the submucous connective tissue and the mucous membrane; the dental sacs rest in depressions in the alveolar process of the jaw. The tooth grows in length mainly as the result of the calcification of its roots, and being thus fixed below, it pushes upward towards the mucous membrane. This growth undoubtedly goes on steadily from birth until the tooth pierces the gum.

The deciduous or milk teeth are twenty in number. The time at which they appear is subject to considerable variation even under normal conditions. The following is the order and the average time of appearance of the different teeth:

|   |                |
|---|----------------|
| (1) Two lower central incisors . . . . .                          | 6 to 9 months. |
| (2) Four upper incisors . . . . .                                 | 8 " 12 "       |
| (3) Two lower lateral incisors and four anterior molars . . . . . | 12 " 15 "      |
| (4) Four canines . . . . .  | 18 " 24 "      |
| (5) Four posterior molars . . . . .                               | 24 " 30 "      |
| At 1 year a child should have . . . . .                           | 6 teeth.       |
| At 1½ " " " " . . . . .   | 12 "           |
| At 2 years: " " " " . . . . .                                     | 16 "           |
| At 2½ " " " " . . . . .   | 20 "           |



Quite wide variations on both sides of the average are common, and are not always easy of explanation. In many cases it seems to be a family idiosyncrasy, since in the different members of a family the teeth are apt to appear at about the same time. The order in which the teeth appear is much more regular than the time of their appearance. Slight variations are exceedingly common, but marked irregularities in the order of the appearance of the teeth are the rule in idiotic children or those suffering from slighter mental defects.

The teeth may pierce the gum without any local manifestations. Very frequently, however, just before a tooth comes through there is noticed a moderate swelling and redness of the mucous membrane of the gum overlying it, and to a slight degree this may affect the general mucous membrane of the mouth. This condition may be accompanied by a little fretfulness and increased salivation, or both of these may be entirely wanting. These symptoms usually disappear when the tooth has pierced the gum. The symptoms of difficult dentition will be discussed in connection with Diseases of the Mouth.

Infants may be born with teeth. I know of one family in which this occurred in three members of three successive generations. It is, however, rare. It is almost invariably one of the lower central incisors that is present. In case this interferes with nursing, or if it is very loosely attached to the gum, it should be extracted, but under other circumstances it should be allowed to remain, since, if it is removed, a second tooth is not likely to appear in its place in the first set. It is not at all uncommon for the first teeth to appear in the fourth month. Such teeth, in my experience, do not usually differ in character from those appearing later, unless they are in children who are syphilitic. Syphilitic children are rather prone to early dentition, and under such circumstances rapid and early decay is likely to take place. Nursing infants are, as a rule, a little earlier in their dentition than those artificially fed.

Delayed dentition is usually due to rickets. However, in many healthy infants no teeth appear before the tenth month; and I have occasionally seen the first ones at thirteen months in those who seemed perfectly healthy and showed no other evidence of rickets. On the other hand, it is by no means invariable that dentition is late in rachitic children. The latest dentition is seen in cases of cretinism. In such children it is not rare for the first teeth to appear as late as eighteen months or two years. As a rule, dentition and ossification of the bones of the head go on in a corresponding manner; where one is early the other is likely to be rapid, and conversely. Great irregularities in dentition are common in children with defective cerebral development.

Provided an infant is well nourished and thrives properly for the first six or eight months, the eruption of the teeth is likely to go on

steadily after this time, even though the child may later have chronic indigestion or suffer from extreme malnutrition from any cause excepting rickets. If, however, the symptoms of malnutrition date from birth, dentition is almost invariably delayed. It is often a matter of very great surprise to see children who are markedly emaciated as a result of chronic indigestion or ileo-colitis and yet go on cutting their teeth regularly. I once had under my care a delicate infant of sixteen months, whose body length was twenty-eight inches and whose weight was less than nineteen pounds—almost exactly what they were eight months previously—and yet he had thirteen teeth.

**Eruption of the Permanent Teeth.**—The first to appear are the first molars, which usually come in the sixth year, and hence the name six-year-old molars, which is applied to them. These appear posterior to the second molars of the first set. The following table from Forchheimer gives the average time of the appearance of the second teeth:

|                         |           |
|-------------------------|-----------|
| First molars . . . . .  | 6 years.  |
| Incisors . . . . .      | 7 to 8 "  |
| Bicuspid . . . . .      | 9 " 10 "  |
| Canines . . . . .       | 12 " 14 " |
| Second molars . . . . . | 12 " 15 " |
| Third molars . . . . .  | 17 " 25 " |

The incisors and canines replace the corresponding teeth of the first set. The eight bicuspid take the place of the eight molars of the first set. The molars of the permanent set appear back of the bicuspid, room being made for them by the growth of the jaw. As they grow and push upward they cause atrophy of the roots of the first teeth, and gradually cut off their blood supply, so that they loosen and fall out.

The place of dentition as an etiological factor in the diseases of infancy will be considered in the chapter on Difficult Dentition.

### CHAPTER III.

#### PECULIARITIES OF DISEASE IN CHILDREN.

IN many particulars disease in children differs from that of later life. These differences relate to etiology, pathology, symptomatology, diagnosis, and prognosis. The greatest contrast to adult life is presented by infancy and early childhood. After seven years, children in their diseases resemble adults more than they do infants.

#### ETIOLOGY.

1. **Inheritance** is an important factor. The disease most frequently transmitted directly is syphilis. Occasionally tuberculosis and other

infectious diseases have been conveyed directly from the mother to the child. In cases where no distinct disease is transmitted, children may inherit from parents constitutional weaknesses or tendencies, which may manifest themselves in infancy, or in some cases not until later childhood. Under this head we may place the influence of alcoholism, lead poisoning, rheumatism, gout, epilepsy, and insanity.

2. **Malformations** must be considered, particularly in the first two years of life. The most important of these, from a medical standpoint, are those of the heart, brain, stomach and intestines, and kidney. The various malformations of the mouth, nose, bladder, rectum, and genital organs belong more particularly to the domain of surgery.

3. **The Diseases or Accidents Connected with Birth.**—Some of these are distinctly traumatic, like the meningeal hæmorrhages. A very large class are the infectious processes in the newly born. Infection usually takes place through the umbilical wound, more rarely through the skin or mucous membranes. This class includes pyæmia, with its varied lesions in the brain, lungs, and serous membranes, erysipelas, ophthalmia, and tetanus. In the class of infectious diseases may also be included many of the varieties of pulmonary and intestinal diseases in the newly born, and probably also some of the hæmorrhagic affections.

4. **Conditions Interfering with Proper Growth and Development.**—These are among the largest etiological factors in the diseases of infancy. They are improper food or feeding, unhygienic surroundings, and neglect. These may cause specific diseases, like rickets or scurvy, or may lead to a condition of general malnutrition or marasmus. In this way they become most important predisposing factors, in infancy, to the acute diseases of the gastro-enteric tract, and later in childhood, to functional nervous diseases.

5. **Infection.**—This has already been mentioned as an important factor in diseases of the newly born. The number of diseases in later life directly traceable to this is very large. Under this head should be included not only the well-known classes of infectious and contagious diseases, but also a very large number of varieties of infection which as yet have not been differentiated, and the nature of which is but imperfectly understood.

#### SYMPTOMATOLOGY AND DIAGNOSIS.

In older children the symptoms of disease are very much the same as in adults, and similar methods of examination may be employed. What is really peculiar to children belongs especially to the first three years of life, before speech has developed. During this period the chief and almost the sole reliance of the physician must be upon the objective signs of the disease. It is not so much that diseases in early life are peculiar, as that the patients themselves are peculiar.

Two fundamental facts are always to be kept in mind: First, that the

common pathological processes are comparatively few, being chiefly of the gastro-enteric tract, the lungs, and the brain, but that the variations in clinical types are almost endless; the second is, that in infants, on account of the susceptibility of the nervous system, functional derangements are often accompanied by very grave symptoms, and may even prove fatal in twelve or twenty-four hours, or there may be speedy and complete recovery after very alarming symptoms. In many of these cases the symptoms are so indefinite that an exact diagnosis is impossible during life, and even the autopsy may throw but little light upon them.

At the bedside it is of great assistance to the physician if he can keep in mind the most frequent forms of acute disease that are likely to be met with. In the first group, including those which are very common, may be placed acute indigestion and ileo-colitis, bronchitis, pneumonia, pharyngitis, and tonsillitis; in the second group, including those which are less frequent, may be placed otitis and the more common acute infectious diseases; in the third group, including the rarer forms of acute disease—meningitis, tuberculosis, rheumatism, and diseases of the kidneys. Under all circumstances, the season, and the nature of the prevailing epidemic, if one exists, are to be considered.

In the examination of a sick infant quite a different method is to be followed from that pursued with adults. Much information is to be gained from a history carefully taken from an intelligent mother or nurse, and much more from a close observation of the child, whether asleep or awake, quiet or crying.

**The History.**—In view of the fact that but little information can be had from the patient, none at all in most cases, it is important to obtain from the mother or nurse as full and complete information as possible. A good history carefully obtained, puts the physician in possession of a fund of information about the patient which is not only of the greatest value in arriving at a diagnosis in the illness for which he is consulted, but is exceedingly helpful in the future management of the child. He may thus know the individual peculiarities and special pathological tendencies. The laity attach great importance, and justly so, to advice from the physician who "knows the child's constitution." Such a history should be taken at the first opportunity after the physician is placed in charge of a child, and with note book in hand, or half its value will be lost.

**Family History.**—This should begin with the parents, going farther back, if possible, in many cases of hereditary disease. One must know regarding tuberculosis, syphilis, rheumatism, or alcoholism, the general vigour of constitution and physical condition of both father and mother. Health during pregnancy and previous miscarriages, if any, are important facts in the mother's history. One should know the number of other children living and their general health, the number dead and

from what causes. A knowledge of the surroundings in which the child has lived may be necessary to appreciate the chances of exposure to tuberculosis, malaria, and many other forms of infection.

*Patient's Previous History.*—This should begin with birth. One should inquire whether the child was premature or born at term, regarding the character of the labour, whether natural or instrumental, tedious or complicated, the condition and vigour of the child at birth, primary respirations, early convulsions, and the nutrition during the early days. Next the methods of feeding should be taken up—how long entirely by and how long partly breast fed, the date of weaning and the form of artificial feeding then employed. If the patient is an infant, and the problem presented is one of its nutrition, all the reliable data relating to the feeding should be obtained, even to the minutest detail. This may be wearisome and consume time, but in no other way can one appreciate the conditions present. The best idea of the child's growth and development may be obtained from a weight record if one has been kept. If not available, one must depend upon general statements as to how the child thrived at different periods. The date of the appearance of the first teeth and the time and the order in which the teeth came, are significant. The general muscular development may be best determined by learning when the child could first hold the head erect, sit alone upon the floor, bear the weight upon the feet, creep or walk alone; the mental development, by learning as to early recognition of mother or nurse, knowing the bottle, understanding the meaning of words, speaking in words or sentences. The muscular and mental development of a normal child during the first two years is a subject with which the physician should be familiar if he would detect early those differences, often slight at this age, in children whose development is backward owing to cerebral lesions.

All previous attacks of acute illness of whatever character should be noted, particularly the infectious diseases—measles, scarlet fever, diphtheria, pertussis, and influenza—with dates and details as to duration, severity, and complications. One should learn whether the child is especially prone to disorders of digestion or those of the respiratory system. Under the former head are included early difficulties in feeding, acute attacks of indigestion, diarrhœa, or dysentery, also chronic disturbances of the stomach or bowels; under the latter head, frequent catarrhal colds, earache or otitis, catarrhal croup, bronchitis, pneumonia, or pleurisy. Other points to be investigated relate to attacks of tonsillitis, operations for the removal of hypertrophied tonsils or adenoids, and previous disorders of the nervous system. In infants, particularly important are extreme restlessness, insomnia, convulsions, or attacks of night terrors; in those who are older, hysterical manifestations, epilepsy, or chorea. Finally, one should know the date of successful vaccination.

Inquiry should also be made concerning any recent exposure to infection in the community, school, or home.

*Present Illness.*—One should first note the chief complaints as stated by mother or nurse. It is important to obtain as definite statements as possible as to the time when the child was quite well, and whether the onset of the illness was abrupt or gradual, and with what particular symptoms. In all digestive disorders one should know exactly concerning the child's food at the time of the onset, its quantity, character, and preparation; also any recent change in diet, the presence or absence of vomiting, and the condition of the bowels, whether loose or constipated, the frequency and character of the stools. General questions as to whether the bowels are regular or the stools normal are of no value, since the informant often is not capable of judging correctly.

Nervous symptoms, like the others, should be elicited in response to direct questions regarding sleep, restlessness, moaning, crying out, or other evidences of pain, excitement, delirium, or convulsions, or unnatural drowsiness. In any acute illness other important symptoms are fever, sweating, dyspnoea, cough, hoarseness, nasal discharge, and the amount and character of the urine.

*The Examination.*—With infants, quite a different method should be followed from that pursued with adults. It may well begin with:

*General Inspection.*—What is learned in this way will depend almost entirely upon the acuteness of observation of the physician, but much that is of value can be ascertained before the clothing is removed for the physical examination by simply watching the patient, whether asleep or awake, for several minutes. In acute disease, the following points should be noted especially:

1. Nutrition and general development: whether the child is well nourished or the features pinched and wasted.
2. The facial expression: whether it is bright and intelligent or dull and stupid, peaceful or anxious, quiet or disturbed, and whether the features are contracted from time to time, as if from pain.
3. The character of the respiration: whether it is rapid or slow, easy or difficult; whether there is nasal obstruction, as indicated by snoring and mouth-breathing, suggesting in infants acute rhinitis, syphilis, or retro-pharyngeal abscess; in older children, diphtheria, scarlet fever, or adenoids. Marked dyspnoea is usually accompanied by active dilatation of the *alæ nasi*.
4. The posture: whether the child lies upon the back, side, or face; whether the head is drawn back with general flexion of the extremities as in meningitis.
5. The nervous condition: whether the child is restless, excitable, or drowsy and apathetic; if asleep, the nature of the sleep should be observed.

6. The colour of the skin of the face: whether pale or cyanotic; and the lips, whether fissured or excoriated.

7. The amount of prostration: a practised eye can usually tell with older children whether the condition is grave or not, but infants not infrequently deceive even the most experienced observer.

8. The cry: in conditions of restlessness or irritability, much information may be obtained from its character. It is important, but not always easy, to determine whether a child cries from fright, as at the approach of a stranger, from nervousness or bad training, from general irritability which may come from any acute disease, or from actual pain. The cry of fright is usually evident, because it comes with the physician's approach and ceases when he goes away. Children of highly neurotic parents and those who have been much indulged and badly trained will often cry when anything out of the usual routine occurs. The cry of pain may be very distinctive; it may be sharp and acute and accompanied by some attempt at localisation, as when a child puts his hand to an inflamed part, but in infancy the pain of acute inflammation is often indicated only by general restlessness and irritability. This is frequently true of acute otitis. The cry of pain is usually accompanied by contraction of the features and other evidences of distress.

The cry of some diseases is quite characteristic, as the short, catchy cry of acute pneumonia or bronchitis; the hoarse cry of laryngitis, whether catarrhal, membranous, or syphilitic; the feeble whine of extreme exhaustion or marasmus; the moaning cry of intestinal disease; and the sharp cry of a child with scurvy whenever its bed or body is touched.

*Measurements.*—These, though of greatest value in chronic diseases, particularly disturbances of nutrition, may be of assistance also in acute conditions. The important measurements are the circumference of the head, chest, and body length. The circumference of the abdomen is at times important, but varies so much with the degree of distention that it is not significant as to the general development. The measurements and weight furnish reliable data which are not only of assistance in the diagnosis of existing disease, but if recorded are useful for future comparison.

In taking the circumference of the head the largest measurement (over the occipital and frontal eminences) is preferable. The measurement of the chest is usually taken over the nipples. The body length of infants is best taken with a tape as the child lies upon his back upon a table or a firm bed. For older children a special measuring stick is convenient.

To estimate properly the significance of measurements they should be compared with the normal averages and with each other. It should be remembered that the head is normally larger than the chest until near

the end of the second year; after this time, with a normal development, the chest should be larger. Any great disproportion between the size of the head and chest is suggestive of disease. The large head and the small chest belong especially to rickets. The measurements form important means of recognising early such abnormalities as cretinism and achondroplasia, the variations often being marked before the other symptoms are prominent. One who forms the habit of taking regular measurements soon appreciates the variations from the normal, and gains great assistance from these data. Such a record made from year to year in children whose development is in any way abnormal is of great value in indicating what should be done in the way of exercise to correct faulty conditions.

*Vital Signs.*—Pulse, Respiration, and Temperature.—The significance of these signs is not to be measured by adult standards, since the susceptible nervous system of infants and very young children greatly exaggerates their reaction to all forms of acute infection.

The rate, regularity, quality, and tension of the pulse should be noted. In young children, the rate of the pulse is of less importance than its force and quality. A slow, irregular pulse is always significant, and should suggest meningitis or brain tumour; an irregular pulse, when rapid, has no special significance. The pulse rate is much increased from slight disturbances; the approach of the stranger or the examination by the physician may cause it to rise 20 or 30 beats. In acute disease, a pulse rate of 150 is common, and 170 or 180 is often seen where other symptoms are not particularly severe.

The rate, depth, and rhythm of respiration should be noted. The last often cannot be determined except by attentively watching the child for several minutes. In premature and very young infants a rather marked irregularity may be seen, often approaching the Cheyne-Stokes type. It is not to be taken as indicating a cerebral lesion, but seems rather to be due to the fact that the respiratory centre is not yet fully able to control the movements. Respiration of this type is seen only during the first weeks of life. Irregularity of rhythm at other times should suggest cerebral disease, usually meningitis. The respiration rate is proportionately greater in infants than in adults. In acute diseases of the lungs it not infrequently rises to 70 or 80, and occasionally it may be over 100 a minute. The rate is generally in proportion to the extent of the pulmonary lesion.

The temperature of infants and very young children should be taken in the rectum, since groin or axillary temperatures are untrustworthy and those in the mouth difficult to obtain. Immediately after birth the temperature of the child is about the same as that of the mother, or a little higher. It falls from 1° to 3° F. in the course of the first few hours. Soon it again rises to 98.5° or 99° F.



From a large number of personal observations upon healthy infants, I have found that the rectal temperature under normal conditions varies between 98° and 99.5° F.; occasionally the range may be as wide as 97.5° to 100.5° F. in apparently perfect health. The heat-regulating centre in the brain acts only imperfectly in the young infant, and slight causes are enough to disturb the temperature.

The temperature in infants is always higher than from corresponding causes in adults. Moreover, very high temperatures may be met with in cases not serious, and not infrequently when no explanation can be found even after thorough examination. In such cases the temperature seldom remains at a high point for more than a few hours. It is a continuous high temperature rather than a single rise which is significant of disease in infancy. Nothing is more perplexing to the young practitioner than the frequency with which a high temperature is seen in infants in cases of comparatively mild illness.

It is common in chronic wasting diseases, in delicate infants and in those prematurely born, to find the temperature one or two degrees below the normal; 95° and 96° F. are of almost daily occurrence in hospitals, and much lower ones are not rare. Daily observations should be made with the thermometer in such conditions, just as in fever.

Puzzling and apparently alarming temperatures are seen in infants as a result of the application of artificial heat. In one of my patients, an infant two days old, a temperature of 107° F. was caused by the close proximity of two large hot-water bags placed in the baby's basket. The younger and feebler the child the more readily are such temperatures produced.

*Muscular and Mental Development.*—The general muscular development is determined by seeing how well the child can hold up his head, sit alone, stand, or walk; the mental development in young infants by the intelligence of expression, the manner in which they respond to stimuli, the recognition of objects, fright at strangers, etc.; later in the first year, by the use of their hands, their understanding of speech, and their ability to pronounce words.

*Local Examination.*—For the purpose of making a complete routine examination of an infant the entire clothing, with the exception of the napkin, should be removed, and the infant placed preferably upon the nurse's lap upon a blanket. With older children the clothing may be removed and the body examined, one part at a time, but with all children it is essential that the examination be complete. A warm room is indispensable, and a table covered with a blanket in many respects better than the nurse's lap, although the latter has usually to be employed. The local examination should be deliberate, the physician should proceed cautiously, winning the child by gradual approaches, and avoiding excitement, force, or anything which may cause pain.

*Skin.*—The skin should first be inspected for eruptions, and it is important that the entire eruption be examined in order that the distribution as well as the character of the lesion may be seen. It should be noted also whether the skin is dry or moist. Marked wrinkling or loss of elasticity of the skin is one of the best indications of loss in weight. Bedsores are more frequently seen over the occiput than over the sacrum, and any large veins should be noted.

External glands should now be examined, especially the cervical, axillary, inguinal, and epitrochlear. The cause of a marked enlargement of any of these groups should be sought in the skin or mucous membranes with which they are connected. Marked swelling of the cervical glands may indicate diphtheria, scarlet fever, or a simple acute inflammation dependent upon a rhino-pharyngitis. Enlargement of the epitrochlear glands is especially significant of syphilis. General enlargement of all the glands to a slight degree is seen in most cases of malnutrition and in many acute infectious diseases.

*Head.*—One should first note whether the sutures are ossified, unnaturally open or separated; also whether the fontanel is closed or, if open, whether it is depressed or bulging. Prominences of the frontal or parietal regions when symmetrical are indicative of rickets. Irregular prominences of a smaller size, when present, are usually syphilitic. In the newly born, a tumour on the head, if in the median line, may indicate an encephalocele; if limited to either the parietal or occipital bone it is usually a cephalhæmatoma.

*Eyes.*—The condition of the conjunctiva and lids should be noted, also the presence of ptosis, strabismus, or other paralysis, but particularly the condition of the pupils, whether contracted or dilated, and the nature of their response to light. One should look also for the presence of corneal ulcers or the interstitial keratitis so frequent in late hereditary syphilis.

*Ears.*—The presence of a discharge may be recognised by sight or by the odour. In any acute febrile condition one should look for tenderness or swelling over the ear or mastoid.

*Nose.*—The presence of any nasal discharge should be noted and its character determined. An abundant discharge tinged with blood, in young infants, should suggest syphilis; in older children, diphtheria; a chronic discharge, adenoid growths; a purulent discharge of one side, a foreign body.

*Mouth.*—The appearance of the mucous membrane of the mouth and gums as well as the teeth may often be ascertained by watching the child while he is crying. It should be noted whether the tongue is dry or moist, clean or coated; whether thrush is present or any other form of stomatitis. If the gums are congested, swollen, or hæmorrhagic, they should suggest scurvy. The number, position, and character of

the teeth are important. The general colour of the mucous membrane may be significant in cases of cyanosis in congenital cardiac disease, and extreme pallor of the mucous membrane in anæmia. On the mucous membrane of the hard palate may often be found the first local evidence of scarlet fever in the form of a minute punctate eruption, and on that portion of the cheeks opposite the molar teeth should be sought Koplik's sign, the earliest reliable symptom of measles.

*Throat.*—A careful examination of the pharynx and tonsils should never be omitted in any acute illness, no matter what other symptoms may be present. Not only tonsillitis, but often diphtheria, is overlooked from a failure to observe this as an invariable rule. A good light is essential, and one must train himself to take in all the appearances at a single glance. Marked general redness of the pharynx may be associated with scarlet fever, influenza, or simple acute pharyngitis. If other symptoms are present pointing to chronic nasal obstruction or to a catarrhal process of the rhino-pharynx, a digital examination should be made to determine the presence of adenoids. Dyspnoea with mouth-breathing when associated with difficulty in swallowing should, in an infant, always suggest retropharyngeal abscess. The examination of the mouth and throat may wisely be made the last step, since it usually disturbs a child so as to embarrass further investigation.

*Neck.*—One should consider the position in which the head is held and the amount of rigidity of the cervical muscles. Opisthotonus may be associated with meningitis or old cerebral palsy. A marked rigidity may indicate cervical Pott's disease or, if accompanied by torticollis, may be of rheumatic origin.

*Chest.*—In young children particular importance should be attached to the shape of the chest. Symmetrical deformities are usually due to rickets. Contraction of one side only is most frequently the result of an old empyema or an extensive interstitial pneumonia. Bulging of the precordial region is frequent in cardiac disease. One should notice also the recession of the soft parts—intercostal spaces, the suprasternal notch, or the epigastrium; the amount of this is usually the best means of judging the severity of obstructive dyspnoea. Details regarding the physical examination of the lungs are discussed in the introductory chapter to pulmonary diseases.

*Heart.*—It should be remembered that under two years old loud murmurs are almost invariably of congenital origin, that soft murmurs at the base are very frequently functional, and that acquired cardiac disease is rare until after three years. For further details in the examination the reader is referred to the chapters upon diseases of the heart.

*Abdomen.*—There should be noted the presence or absence of tympanites or abdominal tenderness, whether general or localised, and the existence of retraction of the abdominal walls as in meningitis. The

size and position of the liver and spleen are best determined by palpation. The lower border of the liver is usually slightly below the free border of the ribs. If the spleen can be easily felt below the ribs, it is, as a rule, enlarged. If it can not be felt in a satisfactory examination, it is not sufficiently enlarged to be of any diagnostic importance. In acute disease a large spleen suggests malaria, typhoid, or tuberculosis; in chronic disease, malaria, syphilis, leukæmia, or anæmia.

*Spine.*—The most frequent spinal curves seen in infancy are those due to muscular weakness. These disappear by placing the child in a prone position. Rachitic curvatures are of the same general character, but as they have usually lasted a longer time the spine is less flexible and the curvatures may not entirely disappear by change of posture. An angular deformity or even marked rigidity of the spine should suggest Pott's disease.

*Extremities.*—The colour of the skin and the character of the peripheral circulation should be noted especially in pneumonia, diphtheria, and other diseases attended by weakened heart. Clubbing of the fingers or toes may be due to congenital heart disease or to chronic disease of the lungs. Desquamation of the palms or soles may indicate hereditary syphilis or scarlet fever, even though no other evidence may be present. The finger-nails may give valuable information in hereditary syphilis. In examining the extremities one should note especially the presence of tenderness, flaccidity, or rigidity of muscles, whether the limbs are wasted or plump, and the degree of muscular power; also any abnormal swelling on the shaft or near the extremities of the bones, and, finally, the function of the joints. A general relaxation of the ligaments is common in rickets and paralytic conditions. Flabbiness of the muscles belongs to malnutrition and rickets; rigidity, if chronic, is usually indicative of cerebral palsy. Weakness of special groups, with atrophy and flaccid muscles, suggests poliomyelitis. Acute tenderness of the legs in infants should suggest scurvy. Rachitic deformities are almost invariably bilateral. Tuberculous bone disease affects the epiphyses, while syphilis usually involves the shafts, the only exception to this being the epiphyseal separation which may occur in the first months of life.

The reflexes may be somewhat difficult to obtain in infants and when exaggerated may indicate cerebral palsy, meningitis, or, as in tetany, only an extreme irritability of the nervous centres without organic disease. The plantar reflex of Babinski has little significance in infants, and in older children it is present in many conditions. Kernig's sign is a form of muscular spasm almost invariably present in cerebro-spinal meningitis, but often seen in other diseases.

*Genital Organs.*—Male children should be examined to determine the presence of phimosis or of undescended testicles. Hydrocele of the cord

is a frequent condition, and may be mistaken for hernia. Both inguinal and umbilical herniæ are very common. In female children it should be remembered that preputial adhesions may be considered normal, and are seldom the cause of the nervous symptoms attributed to them. Every vaginal discharge is significant, and if purulent should be examined bacteriologically. The great frequency of gonococcus infections is not appreciated, and they may be found when least expected.

The examination is not complete without the inspection of the stools, the chemical and microscopical examination of the urine, and an examination of the blood. All are more fully considered in special chapters.

#### PATHOLOGY.

The pathological processes which result from intra-uterine disease and those which are connected with delivery are peculiar to early life. They have already been referred to in the section on etiology. Of the processes of early life which begin after birth, the first in frequency are those of the mucous membranes resulting from the various forms of irritation and infection. In summer, it is the stomach and intestines which suffer chiefly; in winter, the respiratory tract.

The serous membranes are rarely the seat of primary inflammation. The pleura is seldom the seat of primary disease, but is very often involved secondarily to disease of the lung itself. Affections of the pericardium and peritonæum are quite rare. Meningitis is fairly common, especially the tuberculous form.

Diseases of the lymph nodes (lymphatic glands) play an important part in connection with the acute diseases of the mucous membranes, with many affections of the skin, and even of the viscera. Acute infection tends to excite suppurative inflammation, particularly in infants; a less active process leads to chronic hyperplasia in the mesenteric, mediastinal, and cervical glands, in the tonsils, adenoid tissue of the pharynx, etc. The lymph nodes in the neck and thorax are frequently the earliest seat of tuberculous deposits, and in very many cases they are the foci from which secondary infection of the lungs, brain, or joints may occur.

Of the visceral inflammations<sup>1</sup> those of the lungs are the most common, it being rare to find the lungs normal at autopsy after any acute infectious disease which has lasted a week. Up to the third or fourth year of life the heart usually escapes. In older children it may be involved, as in adults, in the rheumatic diseases. The liver and spleen are not often the seat of organic disease in early life, nor is serious disease

<sup>1</sup> The following table gives in a general way a very good idea of the relative frequency of diseases of the different organs in infancy. It is based upon seven hundred and twenty-six consecutive autopsies in the New York Infant Asylum, extending over a period of eight years during my connection with that institution. More than one half of the autopsies I made personally. Of these children seventy-two per cent were

of the kidney likely to be met with excepting in connection with scarlet fever. Organic disease of the brain itself is rare, as is also organic disease of the spinal cord, with the exception of poliomyelitis. Chronic

under one year, twenty-five per cent between one and two years, and only three per cent were over two years. The institution did not receive infants under one month, hence the absence of lesions peculiar to the newly born:

*Table showing principal lesions in seven hundred and twenty-six consecutive autopsies in the New York Infant Asylum.*

*Lungs:*

|   |     |
|---|-----|
| Pneumonia—Primary . . . . .                               | 139 |
| Complicating other acute infectious diseases . . . . .    | 112 |
| Complicating other conditions . . . . .                   | 71  |
| Noted to be present in . . . . .                          | 322 |
| Pleurisy— No case uncomplicated with disease of lungs.    |     |
| Empyema . . . . .   | 5   |
| Serous pleurisy . . . . .                                 | 1   |
| Dry pleurisy in nearly all the severe cases of pneumonia. |     |
| Atelectasis (congenital) . . . . .                        | 6   |
| Pulmonary abscess (always with pneumonia) . . . . .       | 7   |
| Pulmonary gangrene (always with pneumonia) . . . . .      | 2   |
| Pulmonary tuberculosis . . . . .                          | 56  |

*Mouth:*

|                |   |
|----------------|---|
| Noma . . . . . | 1 |
|----------------|---|

*Peritonæum:*

|  |   |
|--|---|
| Acute peritonitis (localised 2, with acute pneumonia and pleurisy 2) | 4 |
|--|---|

*Kidneys:*

|   |    |
|---|----|
| Acute nephritis (complicating scarlet fever 4, diphtheria 1, pneumonia 4, measles 1, pertussis 1, ileo-colitis 2, pyonephrosis 1, apparently primary 5) . . . . . | 19 |
| Malformations of the kidney . . . . .   | 7  |

*Stomach and Intestines:*

|   |      |
|---|------|
| Acute ileo-colitis, with or without gastritis . . . . .   | 116  |
| Acute gastritis (without intestinal lesions) . . . . .    | None |
| Acute diarrhœal disease (without gross lesions) . . . . . | 72   |
| Intussusception . . . . .                                 | 1    |

*Heart:*

|   |      |
|---|------|
| Pericarditis (all with acute pneumonia) . . . . . | 3    |
| Congenital malformations . . . . .                | 3    |
| Acute or chronic endocarditis . . . . .           | None |

*Brain:*

|   |    |
|---|----|
| Acute meningitis (7 with pneumonia, 2 cerebro-spinal) . . . . . | 14 |
| Tuberculous meningitis . . . . .                                | 11 |
| Acute encephalitis . . . . .                                    | 1  |
| Chronic pachymeningitis . . . . .                               | 5  |
| Chronic simple meningitis . . . . .                             | 1  |
| Chronic hydrocephalus . . . . .                                 | 3  |

There were twenty-six deaths from marasmus without gross lesions.

diseases of the different viscera are decidedly rare, except when resulting from acute processes. Diseases of the bones and joints are common, and of extreme importance. They are usually of tuberculous, less frequently of syphilitic, origin. Diseases of the blood are quite common, but as yet but little understood. New growths are rare. The parts most frequently affected are the kidney and the bones. Disorders of nutrition are extremely common and of great importance, particularly rickets and scurvy.

#### PROGNOSIS AND INFANT MORTALITY.

The younger the patient the worse the prognosis in all the diseases of childhood. This is in consequence of the feeble resistance of the infantile organism to all diseases, particularly those which are of an acute nature. On the other hand, the rapid metabolism of childhood makes it possible for many conditions of an organic nature to disappear with time, or, as the phrase is, to be "outgrown," provided the patient can be so placed that the general nutrition can be carried to the highest point.

The accompanying chart (Plate I) shows the mortality of New York City by months during three consecutive years, representing a total mortality of 128,136.

The following table gives comparative figures for three periods of three years each, and shows the reduction in infant and child mortality which has taken place in the last twenty years:

*Deaths—New York City (Manhattan and Bronx).*

|                         | 1890-1892.   | 1898-1900.   | 1907-1909.     |
|-------------------------|--------------|--------------|----------------|
| Under 1 year . . . . .  | 32,916 = 26% | 29,326 = 24% | 30,626 = 22.5% |
| 1 to 2 years . . . . .  | 10,547 = 8%  | 9,012 = 7%   | 8,298 = 6.0%   |
| 2 " 5 " . . . . .       | 9,794 = 7%   | 7,292 = 6%   | 6,579 = 5.0%   |
| 5 " 15 " . . . . .      | 5,470 = 5%   | 6,922 = 5%   | 4,902 = 3.5%   |
| Over 15 years . . . . . | 69,409 = 54% | 71,024 = 58% | 85,741 = 63.0% |
| Total . . . . .         | 128,136      | 123,576      | 136,146        |

The deaths per 1,000 of population show the same reduction. The curves for the different age periods are indicated in the accompanying chart (Fig. 6).

The reduction in mortality in New York has been chiefly in acute gastro-intestinal diseases, marasmus and debility in infants over three months old, and acute infectious diseases, especially diphtheria. The mortality from certain other causes is increasing, notably, acute respiratory diseases, prematurity and diseases of the newly born.

The only age in which the mortality is increased during the summer





PLATE I.

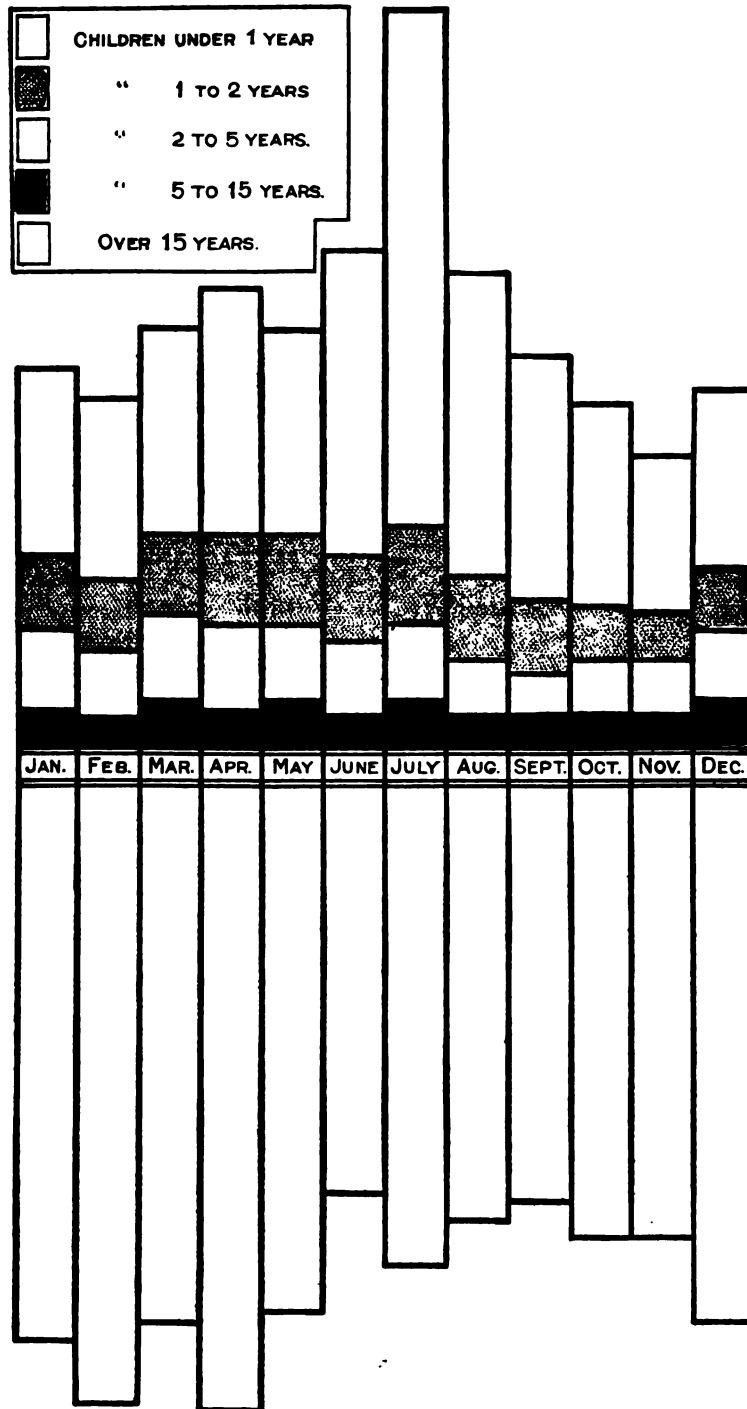


Chart showing by months the mortality of New York city for the different ages for three consecutive years. (Scale, 1 in. = 2,200 deaths.)

months is the first year. In Fig. 7 are given the curves indicating for five years, by months, the deaths under one year and from one to five years.

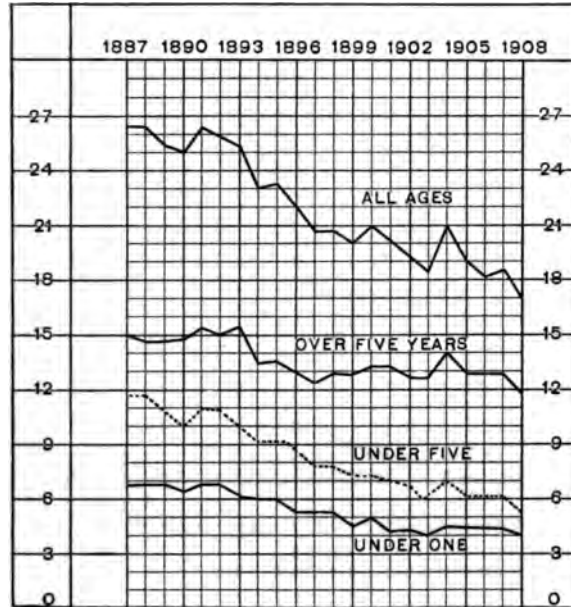


FIG. 6.—DEATHS—NEW YORK CITY PER 1,000 OF POPULATION.

The sharp rise in the summer mortality during the first year is chiefly due to diarrhoeal diseases. It will be noted that the curve for children from one to five years of age touches the highest point in the late winter and early spring months, the time when pneumonia and the

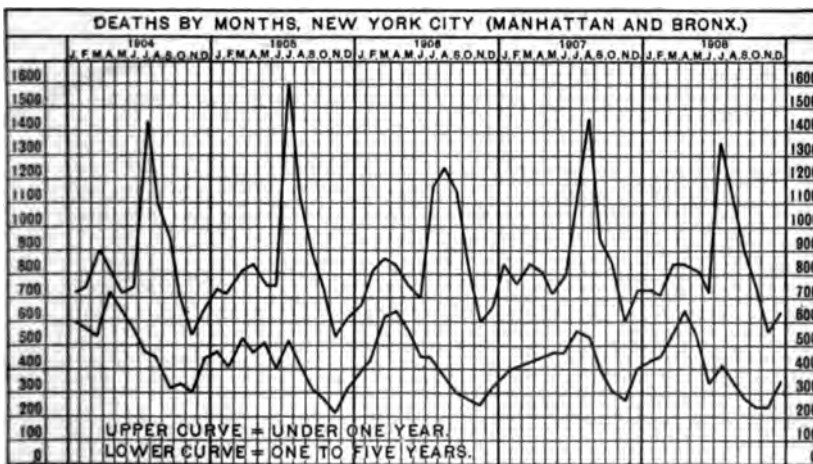


FIG. 7.

common contagious diseases are most prevalent. The curve for both groups is lowest in the months of October and November, which may therefore be considered the healthiest months in New York. The highest mortality is in the first month of age. During this time twenty-five per cent of the deaths of the first year occur. Eröss, writing in 1894, states that from the records of sixteen large cities of Continental Europe nearly ten per cent of all the infants born died during the first month. These figures have been considerably reduced since that time. The first weeks of life are the period of highest mortality, because in them takes place the adaptation of the organism to its environment. After this period each month shows a steadily declining death rate to the end of the first year.

**Causes of Death at Different Periods.**—The most frequent causes of infant mortality, according to the combined reports from the records of

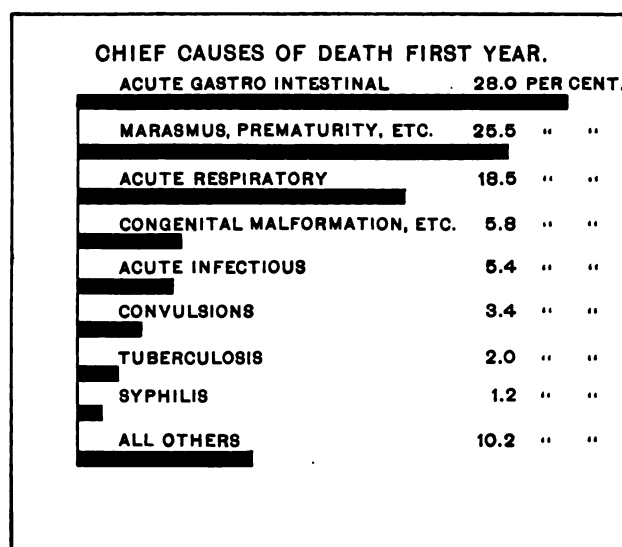


FIG. 8.

the cities of New York, Philadelphia, Boston, and Chicago, making a total of 44,226 deaths in the first year, are shown in the accompanying chart (Fig. 8).

The group, acute gastro-intestinal, includes chiefly diarrhœal diseases in summer. The acute respiratory diseases are pneumonia and bronchitis. Marasmus, prematurity, etc., include also congenital debility, inanition, and other conditions in which the cause of death recorded is disorder of nutrition rather than some general or local disease. The group, congenital malformations, includes also deaths from accidents during birth. Whooping cough is the most important member of

the group of acute infectious diseases, diphtheria coming next. Tuberculosis should, I believe, be rated higher than is shown in these figures. The mortality records of the Babies' Hospital show that the deaths from tuberculosis constitute 5.6 per cent of the first-year mortality of that institution.

The figures and charts above given indicate that a very marked reduction in infant and child mortality has taken place especially within the last twenty years. Many causes have united to bring about this result. Among those which have affected infants may be mentioned: A wider diffusion of knowledge of infant-feeding and hygiene; a great improvement in the general milk supply; the furnishing of pure, whole milk and of modified milk gratis, or at small cost, from milk depots; a general adoption during hot weather of some form of milk sterilisation; the sending of a large number of infants into the country in summer; the closer supervision of infants in cities during the summer by visiting physicians and nurses, and the operation of many other agencies to improve sanitary conditions. Besides these important factors in preventing disease there must be considered the recent advances in pædiatrics and the more rational treatment of the sick infant by the average physician.

During the second year the diseases of the gastro-intestinal tract are still a large factor in the death rate, also the acute diseases of the lungs and the acute infectious diseases, especially measles, diphtheria, and pertussis. Deaths from scarlet fever are much less numerous. General tuberculosis and tuberculous meningitis are frequent.

From the second to the fifth year the deaths are mainly from acute infectious diseases—chiefly diphtheria and scarlet fever—much less frequently from measles or pertussis. In the next group come the acute diseases of the lungs, general tuberculosis, and tuberculous meningitis.

From the fifth to the fifteenth year the mortality in childhood is remarkably small, diphtheria and scarlet fever being still in the front rank in point of frequency. Next come the acute diseases of the lungs, meningitis, diseases of the bones, appendicitis, rheumatism, and cardiac disease.

By far the largest single factor in reducing mortality after the first year is without doubt the use of diphtheria antitoxin. The serum treatment of cerebro-spinal meningitis is important, but not influential in vital statistics, as cases are relatively infrequent.

**Sudden Death.**—This is not a very uncommon occurrence in infants who are apparently healthy. They are sometimes found dead in bed under circumstances in which grave suspicion may unjustly rest upon the attendants. This usually happens with those who are delicate or suffering from malnutrition, especially in institutions where sudden death is by no means rare. The most frequent causes in infants are the following:

1. *Malformations*.—While in most cases malformations of a serious nature give rise to symptoms, they may be absent, or may be so slight as to be overlooked. Infants may succumb during the first few days of life from malformations of the heart, lungs, kidneys, stomach or intestines, and sometimes from diaphragmatic or umbilical hernia.

2. *Internal Hæmorrhage*.—This is chiefly limited to the first two weeks of life. In the cases that have come to my notice the cause has been rupture of some subperitoneal hæmorrhage into the general abdominal cavity, or meningeal hæmorrhage. Such cases are reported in the chapter upon Visceral Hæmorrhages in the Newly Born. Under these circumstances no symptoms may exist until the occurrence of collapse, with death in a few hours.

3. *Asphyxia from Overlying*.—This is not very common, excepting among the lower classes, and is most frequently due to intoxication on the part of the mother. Such infants after death present the usual lesions of death from asphyxia, but without any evidence of violence. A recent writer in the British Medical Journal states that one thousand infants die every year from this cause in the city of London alone.

4. *Asphyxia from Aspiration of Food into the Larynx or Trachea*.—This may be due to vomiting or to the regurgitation of food during sleep; in a very weak infant it may occur while awake. This is usually seen in infants who are less than a year old, and most of the reported cases have been under six months. Such children are usually delicate. There seems to be vomiting with an attempt at crying, during which the food is drawn into the air passages. In some cases, as that reported by Demme, a single large curd of milk has been found in the larynx. In others, food is found in the larynx, trachea, and large bronchi. Cases have also been reported by Partridge and Parrot, and I have myself met with at least three. The infants have generally been found dead in bed within a few hours after feeding. This accident is more likely to happen when an infant lies upon his back.

5. *Enlargement of the Thymus*.—Although these cases are very imperfectly understood, they are not rare. I see two or three each year. The condition is most frequent in infancy, but is not confined to this period. When a child is suffering from some minor illness, often bronchitis, severe attacks of asphyxia may develop and sometimes convulsions may unexpectedly occur and death soon follow. Or the first attack may not be fatal. Sometimes sudden death follows the administration of an anæsthetic, particularly chloroform. In most cases there is found besides an enlarged thymus, a general hyperplasia of the lymphatic tissues throughout the body known as *status lymphaticus*, more fully discussed elsewhere.

6. *Atelectasis*.—In very young infants there may be no symptoms excepting malnutrition until sudden death occurs, sometimes with convulsions and sometimes without any such symptoms. (See Atelectasis.)

7. *Marasmus*.—In this class of cases sudden death is of very common occurrence. These children are often as well two or three hours before death as for several weeks. Death frequently occurs at night, the children being found dead in bed in the morning. In some of the cases the exciting cause seems to be the lowering of the temperature, while in many no exciting cause can be found; the vital spark simply goes out after burning for some time with a feeble intensity. In some of these cases the autopsy reveals atelectasis, but in many cases nothing abnormal is found, death apparently resulting from heart failure.

8. *Convulsions in Children Previously Showing no Special Signs of Disease*.—Many of these cases are seen in children who were previously rachitic; some are associated with the status lymphaticus, and many are manifestations of tetany. The autopsy may show no lesion except cerebral hyperæmia. It is extremely rare for cerebral hæmorrhage to produce death in this way. In some rachitic cases death is due to spasm of the glottis.

9. *Asphyxia in Older Infants and Young Children*.—This may result from the pressure of a retropharyngeal abscess upon the larynx or trachea, or from the rupture of such an abscess into the air passages. Previous symptoms may have been wanting. Pressure upon the pneumogastric nerve leading to fatal asphyxia may be caused by tuberculous bronchial nodes, or by abscesses in the posterior mediastinum connected with caries of the spine. Sudden death may occur with spinal caries from dislocation of the upper cervical vertebræ.

Sudden asphyxia may follow the ulceration of tuberculous lymph nodes and the escape of cheesy masses into the trachea or primary bronchi. This usually occurs in children from two to five years old.

10. *Death after a Few Hours' Illness, in which the Chief Symptom is High Temperature*.—This is not an uncommon occurrence. Infants apparently well may be taken with great prostration and a high temperature, which may rise rapidly to 106° or even 107° F., and death follow in from six to twelve hours, sometimes preceded by convulsions. These are often examples of acute septicæmia, most frequently from the pneumococcus, sometimes from the streptococcus, or other organisms. In older children death may be due to malignant scarlet fever or epidemic meningitis; however, unless these diseases are prevailing epidemically, it is somewhat hazardous to make such a diagnosis.

It does not fall within the scope of this chapter to consider cases of sudden death from heart failure after diphtheria, with pleurisy with effusion, or with myocarditis. These will be discussed elsewhere.

#### PROPHYLAXIS.

There is no more promising field in medicine than the prevention of disease in childhood. The majority of the ailments from which children

dic it is within the power of man in great measure to prevent. Prophylaxis should aim at the solution of two distinct problems: (1) The removal of the causes which interfere with the proper growth and development of children; (2) the prevention of infection. The former can come only through the education first of the profession and then of the general public, in the fundamental principles of infant feeding and hygiene. This is a department which has received altogether too small a place in medical education. The latter must come through the profession, and through legislation, the purpose of which shall be more rigid quarantine, more thorough disinfection, and improved sanitation in all its departments. The subject of prophylaxis will be discussed in connection with the different diseases treated elsewhere.

#### THERAPEUTICS.

Therapeutics in infancy consists in something more than a graduated dosage of drugs. Many therapeutic means which are valuable in adults are useless in children, and many others which are of little value in adults are extremely useful in children. Children in the past have suffered much from overzealous treatment, particularly from drug-giving. In early life more than at any other period the old dictum *non nocere* should be heeded. It should be a fundamental principle never to give a dose of medicine without a clear and definite indication. If this rule is followed, it is surprising to find how often medication can be dispensed with. A second rule is equally important: never to give a nauseous dose when one, that is palatable, will answer the purpose equally well. The simpler prescriptions are made, the better. As a rule, infants revolt against most of the highly seasoned sirups and elixirs which are used to disguise the taste of unpleasant doses. Bitter medicines when mixed with water, are frequently administered without difficulty.

It is a common mistake to underestimate the importance of the hygienic surroundings of the patient, the value of good nursing, careful feeding, and judicious stimulation, just as it is to overestimate the beneficial effects of drugs. In the great majority of acute ailments not serious in character, for which a physician is called, the patient recovers quite as promptly without drugs as with them. This does not mean that such children require no treatment, but that the least important part of the treatment is drug-giving. In cases of severe illness, in infants especially, we must avoid all unnecessary medication, in order that the stomach may not be disturbed. Hence the importance of relying as far as possible upon local measures. The strong tendency to recovery from all acute processes, while seen in adult life, is even more striking in childhood, where, if we can but remove that which hampers the bodily

functions, Nature will usually conduct the case to a satisfactory termination. Thus, after an attack of bronchitis, it is often seen that the disturbance of the stomach and intestines can be directly traced to the drugs employed, and continues long after the original disease has subsided. In diseases of the stomach and intestines especially there is a great amount of unnecessary medication. In all chronic disturbances of nutrition—chronic indigestion, malnutrition, and anæmia—no tonic is so good as a change of air and surroundings.

**Antipyretics.**—The indications for the employment of antipyretics in children are somewhat different from those in adults. It is to be borne in mind that, where the cause is similar, all temperatures in children are higher than in adults. Thus conditions, which in an adult would produce a rise of temperature of only 100° or 101° F., in a child are not infrequently accompanied by a temperature of 104°, or even 105° F. The height of the temperature, as measured by the thermometer, is not to be taken as the only or even the best guide for the employment of antipyretics. The nervous disturbance which accompanies such a temperature is much more important. The temperature may be 104°, or even 105° F., and yet the child exhibit no signs of unusual discomfort. Such a temperature manifestly does not call for interference. High temperature from apparently trivial causes, is very common. It is only a continuously high temperature or a recurring high temperature which indicates serious illness. Whenever the temperature is found to be much above the normal it should be carefully watched, but not interfered with until a diagnosis has been made, unless the symptoms urgently demand it; otherwise the physician may lose one of the most valuable aids to diagnosis, since it is not the height of the temperature but its course which is significant. In many cases it is very important to know whether the temperature uninfluenced by drugs is remittent, intermittent, or steadily high, and hence the advantage of waiting until a diagnosis has been made before disturbing the temperature curve. This is, of course, not admissible when the temperature is itself a source of real danger, which after all is seldom the case. Since the cause of a great many obscure temperatures is found in the stomach and intestines, it very often happens that a purgative, stomach-washing, or intestinal irrigation may be the most efficient antipyretic. In cases of moderate elevation of temperature we need go no further than cold sponging.

The most reliable antipyretic measure for children is the use of cold. This may be employed—

(1) *As an Ice Cap to the Head.*—In many cases of quite high temperature and restlessness in infants this alone will reduce the temperature one or two degrees and allay the nervous symptoms.

(2) *Cold Sponging.*—For this purpose water at about 80° to 85° F., equal parts of alcohol and water, or equal parts of vinegar and water may



be employed. In the case of infants, all the clothing except the diaper should be removed and the child laid upon a blanket. The body should be sponged for from ten to twenty minutes, and then wrapped in a blanket without further dressing. Cold sponging must be very frequently employed in order to be efficient in reducing high temperature. Its great value in allaying nervous symptoms, even when the temperature is not very high, is not sufficiently appreciated. Its effect is often more satisfactory than that of an anodyne.

(3) *Cold Pack*.—This is one of the most efficient means of reducing temperature which can be employed. The child should be stripped and laid upon a blanket. The entire trunk should then be enveloped in a small sheet wrung from water at a temperature of 100° F. Upon the outside of this, ice may now be rubbed over the entire trunk, first in front and then behind. By this method there is no shock and no fright, and any ordinary temperature can usually be readily reduced.

The rubbing with ice should be repeated in from five to thirty minutes, according to circumstances, after which the child may be rolled in the blanket upon which he is lying without the removal of the wet pack. The head should be sponged with cold water while this is being carried on, and artificial heat, if necessary, should be applied to the feet. The pack is continued from one to twenty-four hours, according to circumstances.

(4) *The Cold Bath*.—The child is put into a bath at a temperature of 100° F., the temperature being gradually lowered by the addition of ice or cold water to 85° or 80° F. The body should be well rubbed while the child is in the bath and water should also be applied to the head. On removal from the bath, the body should be quickly dried and rolled in a warm blanket. The bath is usually continued from five to ten minutes.

(5) *Evaporation Baths*.—The trunk is closely enveloped in two layers of surgeon's gauze, or some loosely woven equivalent, which is moistened from time to time with water at a temperature of 115° F., continuous evaporation being kept up by means of a hand, or better electric, fan. The evaporation bath would seem to possess some important advantages in the case of infants and young children, in that it is more efficient than sponging, involves little disturbance of the patient, and causes no shock or fright. Hot applications should constantly be made to the extremities.

(6) *Rectal Irrigations*.—These are easily given, disturb the patient very little, and are effective in reducing the temperature. A double tube or two catheters may be employed. It is best to use at first water at a temperature of 90° F., and gradually reduce this to 70° F. The irrigation should be continued for ten or fifteen minutes, or even longer if the desired fall in temperature is not obtained, and may be repeated as often as every three hours.

**Antipyretic Drugs.**—Except in cases of malaria, quinine should not be employed for the reduction of temperature in children.

Of the many coal-tar derivatives employed, phenacetine has the advantage for children of being tasteless and causing little depression, but the slight disadvantage of practical insolubility. None of the drugs of this group is, however, to be employed in large doses with the sole purpose of reducing the temperature. Their great value in pædiatrics consists rather in allaying the nervous symptoms which accompany fever, and this purpose can be accomplished by the use of comparatively small doses. To an infant of one year, phenacetine or antipyrine can be given in one-grain doses every hour or two hours until the desired effect is produced. For a child of five years a dose of two grains may be given in the same manner. When used as indicated, these drugs are of very great value in making the patient more comfortable, in promoting sleep, and in allaying headache and general pains. In cases of hyperpyrexia they are, however, much less certain and less safe than the use of cold.

**Stimulants.**—In spite of the many statements to the contrary, alcoholic stimulants are well tolerated even by very young infants. All stimulants, and alcohol in particular, are very greatly abused in the hands of practitioners, and their indiscriminate and protracted use can not be too strongly condemned.

The indications for the employment of stimulants are much the same in young children as in adults. In most of the acute fevers they are not to be given early in the disease, and in many cases they are not required at all. They must often be used very sparingly while the temperature is high, but may be necessary as soon as it falls. In many acute febrile diseases stimulants are not called for at any period.

The method of administering alcohol is of no little importance. Brandy and whisky are in most cases to be preferred to the wines, but not always. For infants under one year old, brandy should be diluted with at least eight parts of water. Altogether the best method of administration is to determine the amount to be given in every twenty-four hours, have it diluted sufficiently, and then administer it in small doses at short intervals.

An infant one year old, for whom alcohol is indicated, should not be given to begin with more than one-fourth of an ounce of brandy or whisky during the twenty-four hours, and even in bad conditions it is rarely advisable to give more than twice this quantity, except for a very short period. In children four years old double the amount may be employed in the corresponding conditions. Little good and much harm is likely to follow such amounts as six or eight ounces daily of brandy or whisky for children of two or three years. There certainly is a strong tendency at the present time to use less and less alcohol in therapeutics, and many would abandon it altogether.

Other stimulants, caffeine, camphor, strychnine, digitalis, strophanthus, etc., are used in children with much the same indications as in adults. Their application is more fully discussed in the different diseases in which they are employed. They may be used in the following doses at the different ages indicated:

|  | 3 months.           | 1 year.            | 5 years.           |
|--|---------------------|--------------------|--------------------|
| Digitalis, tincture . . . . .                  | ℥ i                 | ℥ iii              | ℥ v                |
| Strophanthus, tincture . . . . .               | ℥ i                 | ℥ i                | ℥ v                |
| Caffeine citrated . . . . .                    | Gr. $\frac{1}{4}$   | Gr. $\frac{1}{4}$  | Gr. i              |
| Strychnine sulphate . . . . .                  | Gr. $\frac{1}{100}$ | Gr. $\frac{1}{50}$ | Gr. $\frac{1}{25}$ |
| Camphor (10 per cent solution in oil). . . . . | ℥ iii               | ℥ vi               | ℥ x                |
| Adrenalin (1-1000 Sol.) . . . . .              | ℥ i                 | ℥ iii              | ℥ vi               |

NOTE.—Camphor and adrenalin are for hypodermic use only. The dosage of all these stimulants is calculated for administration at intervals of four hours.

**Tonics.**—Cod-liver oil is particularly useful in the convalescence after acute diseases of the respiratory tract, in anæmia, and in a large number of children who are extremely delicate. In these patients it may be advantageously administered throughout the greater part of nearly every winter season. In convalescence after attacks of gastro-enteric disease it should be withheld for a long time. When the tongue is coated, the digestion poor, and the stomach easily disturbed it should not be given at all. In the case of infants, as a rule, the pure oil is to be preferred to the emulsion. The administration of small doses—i. e., ten or twenty drops of the oil three times a day continued for a long period—is much better than the use of larger doses for a shorter time.

Preparations of malt are sometimes of even greater value than cod-liver oil, for they can be used in many conditions when the oil is contra-indicated. The two may often be advantageously combined. The best preparations of iron for very young children are the bitter wine, sweet wine, saccharated carbonate, and the wine of the citrate. These are only slightly constipating, and many of them can be given with milk. Most of the organic preparations of the market are less reliable than those mentioned. For older children nothing is better than reduced iron or Blaud's pills.

Arsenic is second only to iron in the treatment of the anæmia of children, and in very many cases it is to be preferred to iron. The tablet triturates of arsenious acid, one one-hundredth of a grain, may be given immediately after meals three times a day, or one or two drops of Fowler's solution largely diluted with water.

Alcohol is useful in combination with some of the bitters, either small doses of quinine, nux vomica, or the bitter wine of iron. Usually wines, especially sherry, are to be preferred to spirits, although some children take spirits better. When combined with a bitter there is little

danger of the formation of the alcoholic habit, even though its use may be long continued.

Of the bitter tonics, nux vomica is easily superior to all others.

**Opiates.**—Strong objections have been urged by many against the employment of opium in the diseases of infancy. While opiates have no doubt been abused, the fact remains that opium is almost as valuable a remedy in the treatment of disease during the first five years as at any other period of life. For infants relatively smaller doses are required than of most drugs. If the physician will accustom himself to the use of very small doses, he will be surprised to see how satisfactory are the effects produced.

The most useful preparations for young children are paregoric, Dover's powder, the deodorised tincture, morphine, and codeine. The following table gives what may be considered safe initial doses at the different ages:

|                          | 1 month.                      | 3 months.                     | 1 year.                                       | 5 years.                               |
|--------------------------|-------------------------------|-------------------------------|---|--|
| Paregoric.....           | $\mathfrak{M}$ i              | $\mathfrak{M}$ ii             | $\mathfrak{M}$ v to x                         | $\mathfrak{M}$ xxx to xl               |
| Deodorised tincture..... | $\mathfrak{M}$ $\frac{1}{80}$ | $\mathfrak{M}$ $\frac{1}{40}$ | $\mathfrak{M}$ $\frac{1}{4}$ to $\frac{1}{2}$ | $\mathfrak{M}$ ii to iii               |
| Dover's powder.....      | Gr. $\frac{1}{80}$            | Gr. $\frac{1}{40}$            | Gr. $\frac{1}{4}$ to $\frac{1}{2}$            | Gr. ii to iii                          |
| Morphine.....            | Gr. $\frac{1}{6000}$          | Gr. $\frac{1}{3000}$          | Gr. $\frac{1}{2400}$                          | Gr. $\frac{1}{360}$ to $\frac{1}{180}$ |
| Codeine.....             | Gr. $\frac{1}{3600}$          | Gr. $\frac{1}{1800}$          | Gr. $\frac{1}{600}$                           | Gr. $\frac{1}{180}$ to $\frac{1}{90}$  |

Ordinarily doses like the above should not be repeated oftener than every two hours. In exceptional circumstances, as when very great pain is present, the dose may be given more frequently. In the hypodermic use of morphine it should be remembered that its effects are always more uniform and striking than when the drug is administered by the mouth, and the dose should therefore be smaller. In every instance where a full dose of opium has been given the physician should wait until the effects have subsided before the dose is repeated.

**Sedatives.**—For most of the milder conditions where sedatives are required bromides are to be preferred. A preference should be given to the sodium salt. Young children require relatively large doses: e. g., in convulsive conditions three grains every two hours are often necessary at three months. Chloral is usually well borne even by quite young infants. Since it is often irritating to the stomach it may be advantageously given by the rectum. After rectal administration its effects are usually manifest in half an hour, and sometimes sooner. The rectal dose for an infant of one month is one grain; three months, two grains; one year, three to five grains. It may be repeated every two to four hours, according to indications. Doses by mouth should be about half as large. Other drugs may replace this in most diseases, but in the case of infantile convulsions nothing is so reliable as chloral.

Belladonna is well borne by children, and in relatively larger doses than most drugs. A tolerance is quite readily established. The eruption is more readily produced than the other physiological effects, and even quite small doses may be sufficient to bring out a very abundant blush. The parents should be advised of this fact, lest undue alarm be felt.

The drugs classed as antipyretics—phenacetine and antipyrine—are exceedingly valuable in the treatment of many diseases of infancy where irritative nervous symptoms are prominent. In many cases they may advantageously take the place of opium, except where pain is the principal symptom, as in otitis or pleurisy. In all conditions where spasm is a prominent symptom, whether of the larynx or bronchi, or local or general convulsions, antipyrine is especially valuable.

**Drugs Well Borne by Children.**—In this list might be mentioned belladonna, the bromides, the iodides, chloral, quinine, calomel—in fact, all mercurials—and alcohol.

The drugs not well borne include particularly cocaine and heroin. In the case of many others, while the constitutional effects are well tolerated, they must be given carefully to young infants, since they are irritants to the stomach. In this class may be mentioned the salicylates, salol, the astringent preparations of iron, and the acids.

**Vaccines.**—These are suspensions of dead bacteria in a normal salt solution. Their application in pædiatrics is confined to therapeutics; as a prophylactic measure they are seldom called for. Vaccine therapy has been employed in almost every form of bacterial infection. In the great majority of these the results have been disappointing. They are of unquestioned value in localised staphylococcus infections, particularly those of the skin, e.g., general furunculosis and larger multiple abscesses. In other staphylococcus infections they are sometimes useful, but results are very uncertain. In streptococcus infections whether localised or general their effect is doubtful; although in rare cases they have seemed to be of benefit. Pneumococcus infections are apparently not at all influenced by their use. Regarding the effect of vaccines on gonococcus infections of mucous membranes, one must speak very guardedly. The great majority of patients with gonococcus vaginitis so treated have received but temporary benefit, although a few striking cures have been obtained. Colon infections of the urinary tract (pyelitis) sometimes appear to be decidedly improved by vaccines. With respect to most other conditions experience thus far does not warrant one in forming a sanguine opinion of their value. For the technique of vaccine treatment special works should be consulted.

**Counter-irritants.**—These are of great value in a large variety of diseases.

The *mustard paste* is probably the most satisfactory means of producing quick counter-irritation over a large surface. To make a mustard

paste: Take one part powdered mustard and six parts of wheat flour, mix with lukewarm water, and spread between two layers of muslin. This should be removed as soon as a thorough redness of the skin has been produced—in most cases from five to eight minutes, according to the strength of the mustard employed. This may be repeated as often as every three hours, and continued for a week if necessary, without producing excoriations of the skin. For older children the paste may be made one part mustard to four parts flour. In pulmonary diseases it should be large enough to surround the chest. When it is used to produce general reaction in heart failure it should cover the entire trunk.

*The Mustard Pack.*—The child is stripped and laid upon a blanket, and the trunk is surrounded by a large towel or sheet saturated with mustard water. This is made as follows: One tablespoonful of mustard to one quart of tepid water. In this a towel is dipped, and while dripping wound around the entire body. The patient should then be rolled in the blanket. This pack may be continued for ten or fifteen minutes, at the end of which time there will usually be a very decided redness of the whole body. It may be repeated according to indications. Where it is desired to produce a general counter-irritation, the mustard pack is not quite as efficient as the mustard bath, but it has the advantage in causing much less disturbance to the patient. The mustard pack is useful in the condition of collapse or of great prostration from any cause whatever, in convulsions, and in cerebral or pulmonary congestion.

The *turpentine stupe* is made by wringing a piece of flannel out of water as hot as can be borne by the hand. Upon this is sprinkled ten or fifteen drops of the spirits of turpentine. The stupe is then applied to the body and covered with oiled silk or dry flannel. It is useful chiefly in abdominal pains or inflammations, but in infancy must be carefully watched or vesication will be produced. For continuous use it is not so valuable as the mustard paste.

*Stimulating liniments* containing turpentine and other irritants are useful in inflammations of the chest, although less reliable than the mustard paste. One of the mildest and most useful preparations is camphorated oil. Another is olive oil four parts and turpentine one part. These may either be rubbed upon the surface, or a piece of flannel may be saturated with them and then applied to the skin.

*Local Blood-letting.*—Leeches are sometimes useful in the early stages of acute inflammations of the mastoid or middle ear. They may also be applied to the præcordium in acute pneumonia with signs of failure of the right heart, viz., great dyspnoea and cyanosis.

*Dry cups* may be used even in young infants, to relieve acute congestion in pneumonia or bronchitis, and for pulmonary œdema. Wet cups should never be used for young children.

**Poultices** are much too frequently employed and may with advantage be omitted in the treatment of most local inflammations. They have been largely replaced by wet dressings, especially those of aluminum acetate. In acute pulmonary inflammations their use is very limited.

**Cold.**—Cold is useful in almost all forms of local inflammation. In inflammation of the cervical lymph glands and of the joints it is of undoubted value, but its advantage over heat is questionable. The efficiency of both cold and heat in these cases depends largely upon the method of use. The difficulties in the way of their proper application are great in young children. Sometimes in pleurisy much greater relief is obtained from the use of an ice bag to the chest than from hot applications, but this is not the general experience. The treatment of pneumonia by the application of the ice bag to the chest has many advocates, although in my own hands it has not yielded the results claimed for it. It is admissible only in lobar pneumonia, and here chiefly in older and stronger children. The application of cold in young or very delicate children should be made with caution in all inflammations of the respiratory tract.

Cold is best applied to the head by an ice cap made like a helmet; an ordinary rubber or flannel bag filled with ice may answer the purpose. The rubber coil filled with ice water is also an excellent method. For inflamed glands or joints the ice bag or the coil should be used; for the eyes, cold compresses, changed every minute.

**The Hot Pack.**—All clothing is to be removed and the child's body covered with towels wrung from water at a temperature of from 100° to 108° F., after which the body should be rolled in a thick blanket. These hot applications may be changed every twenty or thirty minutes until free perspiration is produced, which may be continued as long as necessary. This is mainly useful in uræmia.

**The hot bath,** like the mustard pack or the mustard bath, may be used to promote reaction in cases of shock or collapse. The patient should be put into the bath at a temperature of 100° F., the water being gradually raised to 103°, or even to 106°, but rarely above this point. The body should be well rubbed while the patient is in the bath. A thermometer should be kept in the water to see that the temperature does not go too high. Unless this precaution is taken the danger of burning the child is great. During the bath, in most cases, cold should be applied to the head.

**The Hot-Air or Vapour Bath.**—All the clothing should be removed and the patient laid upon the bed with the bedclothing raised above the body ten or twelve inches, and sustained by means of a wicker support. The bedclothing should be pinned tightly about the neck, so that only the head is outside. Beneath the bedclothing hot vapour is introduced from a croup kettle or a vapouriser. This will usually induce free per-

spiration in fifteen or twenty minutes. It may be continued from twenty to thirty minutes at a time. Instead of vapour, hot air may be introduced in the same way. The air space about the body is indispensable. The vapour bath is applicable chiefly to cases of uræmia.

**The Mustard Bath.**—Four or five tablespoonfuls of powdered mustard should be mixed for a few minutes with one gallon of tepid water. To this should be added four or five gallons of plain water at a temperature of 100° F. The temperature of the bath may be raised by the addition of hot water to 103° or 106° F., if desired. Nothing is more efficient than the hot mustard bath for a general derivative effect in bringing the blood to the surface in cases of shock, collapse, heart failure from any cause, or in sudden congestion of the lungs or brain. The bath should not usually be continued for more than ten minutes. If necessary, it may be repeated in an hour.

**The Bran Bath.**—Put one quart of ordinary wheat bran in a bag made of coarse muslin or cheese cloth and place this in four or five gallons of water. The bran bag should be frequently squeezed and moved about until the bath water resembles a thin porridge. It may be of any temperature desired, but usually about 90° to 95° F. is best. A bran bath is of great value in cases of eczema, excoriations about the buttocks, or in other cases where the skin is very delicate, and plain water seems to irritate it.

**The tepid bath** may be given at a temperature of 95° to 100° F. It is very useful in many conditions of excitement or extreme nervous irritability. To induce sleep it is often more efficient than drugs.

**The cold sponge or the shower bath** should be given in the morning before breakfast, and in a warm room. The child should stand in a foot tub containing warm water enough to cover the feet, then a large sponge holding about a pint of water at a temperature of from 40° to 60° F. should be squeezed three or four times over the chest, shoulders, and spine of the child, the skin being rubbed meanwhile. The bath should not last more than half a minute. It should be followed by a brisk rubbing until a thorough reaction is established. This is very useful at all ages, but it is a particularly valuable tonic in delicate children. It may be used in those only eighteen months old. Not the least of the beneficial results is the full expansion of the lungs from the strong cry which the bath usually excites. In younger infants a cold plunge may be substituted. This should be merely a single dip of the entire body in water at a temperature of 50° to 60° F. In order that beneficial effects shall follow the cold plunge or cold sponging, a good reaction must be established. If children lack sufficient vitality to secure this, and if they remain pale, pinched, and blue for some time after the bath, it must be discontinued altogether, or water of a higher temperature used.



**Nasal Spray.**—This may be either of an aqueous or oily solution. For the oil spray an atomiser should be employed. It is valuable in cases of dry catarrh, where there is a formation of crusts in the nose. A variety of oils may be used, benzoinol being perhaps as satisfactory as any.

There are many forms of hand atomisers to be found in the market for the production of aqueous or oil sprays. For a cleansing nasal spray, Dobell's solution, Seiler's solution, or a two-per-cent solution of boric acid may be used.

**Nasal Irrigation.**—In cases of considerable nasal obstruction and in the more serious affections of the rhino-pharynx, only the syringe can be considered an efficient means of cleansing the cavity.

The fountain syringe has the advantage of being easily regulated as to the force employed, this being determined by the height at which the bag is suspended above the bed. For ordinary purposes an elevation of one or two feet is sufficient, and rarely is a greater pressure than three feet advisable. The last is desirable when a thorough flushing



FIG. 9.—NASAL SYRINGE.

of the rhino-pharynx is required. The position of the patient is the same as that shown in Fig. 10. The danger of forcing fluid into the middle ear is greatly lessened if the patient keeps the mouth wide open.

Where a smaller amount of fluid is sufficient a piston syringe may be employed. This should be small enough to be easily worked with one hand. It should have a soft rubber tip, to prevent injuring the nasal mucous membrane, and the tip should be large enough to fill the nostril. The best piston syringe for nasal use is shown in Fig. 9. This is made either of glass or hard rubber, and fulfils all the conditions mentioned. It is easy of action, can be readily cleansed, and holds about half an ounce. The same syringe should not be used for more than one patient, unless it has been very thoroughly disinfected. In hospitals, and even in private practice, nasal syringes are frequent carriers of infection.

Either of two positions may be used in nasal syringing. In diphtheria, scarlet fever, or any constitutional disease attended by great depression, the child should not be removed from the bed. The syringing may be done by a single nurse, who stands at the head of the bed, alternately syringing the right and left nostril, turning the head from side

to side (see Fig. 10). The other method is to hold the child erect on the lap, with the head inclined somewhat forward, the syringing being done by a second person standing behind. In either position the child's arms and hands should be securely pinioned to the sides by



FIG. 10.—METHOD OF SYRINGING THE NOSE.

a sheet. To make sure that the rhino-pharynx has been reached the water should return through the opposite nostril or the mouth. When properly done, no prostration and very little irritation are caused. The bulb (Davison) syringe should not be employed for nasal irrigation; as the pressure can not be satisfactorily regulated, fluids are likely to be forced into the Eustachian tubes.

**Syringing the mouth and pharynx** is useful in many pathological conditions of these parts, particularly in children too young to gargle. Either the fountain, piston, or bulb syringe may be used. If the pharynx is to be reached, the nozzle is used as a tongue depressor. This should be placed at the angle of the mouth between the back teeth. The child should lie upon the side or be held in the sitting posture, with the head inclined forward. Only bland solutions should be employed.

**Inhalations.**—These are of very great utility in all affections of the respiratory tract. To be efficient, the patient should be put under a tent. A satisfactory tent may be made by erecting a T-shaped piece of wood at the head and foot of the crib and throwing over this a large sheet folded and pinned at the corners. Another method is, to stretch a cord around the top of each of the four posts of the crib, or simply from the centre of the head piece to the centre of the foot piece; the sheet

should be used as in the first instance. A very good tent may be improvised by throwing a large sheet over an open umbrella. Instead of an ordinary cotton sheet one of rubber cloth may be used. For hospital use I have found it convenient to have a rubber cover made to fit closely over the top of the crib to be used for inhalations. The better the tent the more satisfactory are the results from inhalations.

Inhalations may be in the form of vapour or spray. The apparatus employed may be the croup kettle, the vapouriser, or the steam atomiser. As all of these are used with alcohol lamps, innumerable accidents from fire have occurred with them. Patients and nurses should always be cautioned regarding this. Whenever possible, the electric heater should be substituted. The ordinary croup kettle is a clumsy affair and especially likely to be the cause of accidents. In Fig. 11 is shown one of an improved pattern,<sup>1</sup> which possesses the advantages both of the ordi-



FIG. 11.—THE AUTHOR'S CROUP KETTLE.

nary croup kettle and of the vapouriser. The base has been weighted, to prevent the apparatus being easily upset. The pail is low, which fact also contributes to its stability. It is provided with a safety alcohol lamp, the flame of which can be regulated by a screw. The lamp holds enough alcohol to burn from five to six hours. This kettle may be used to produce simple vapour, or vapour from lime water, or a medicated vapour may be employed. If the latter is desired, the substance to be vapourised is placed on a sponge held in the expansion of the spout. The kettle should be filled with hot water before using. It should be placed upon the floor or a low box beside the crib, standing in a large tin basin to

avoid accident, at such a height that the end of the spout is just inside the tent at a level with the surface of the bed.

There are various other forms of apparatus for the purpose of obtaining medicated inhalations.

**Stomach-washing** consists in the introduction of water into the stomach through a flexible catheter or stomach tube and then siphoning it out. It was introduced into general practice among infants by Epstein, of Prague. It is one of the most valuable therapeutic measures we possess. The procedure is very simple, and may be considered entirely free

<sup>1</sup> Made by Lewis & Conger, 130 W. 42d St., New York.

from danger; in fact, it is difficult to pass the tube anywhere else than into the œsophagus. The amount of prostration produced by stomach-washing may be compared to that of an ordinary attack of vomiting.

The apparatus for stomach-washing (Fig. 12) consists of a soft-rubber catheter, size 16, American scale (24 French)—one with a large eye is preferred; a glass funnel, holding four to six ounces; two feet of rubber tubing, and a few inches of glass tubing to join this to the catheter. The child should be held in a sitting or recumbent posture (Fig. 13), the body well protected by a rubber sheet, with a large basin conveniently near. The catheter should be moistened. While the tongue is depressed with the forefinger of the left hand, the catheter is passed rapidly back into the pharynx and down the œsophagus. It is important that the first part of the introduction should be as rapid as possible, for if the child begins to gag from the pharyngeal irritation the introduction of the tube may be quite difficult. No resistance is ordinarily en-

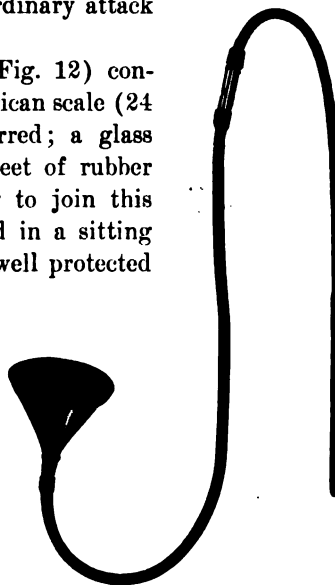


FIG. 12.—APPARATUS FOR STOMACH-WASHING.

countered after the tube reaches the œsophagus. About ten inches of the catheter should be passed beyond the lips. When it has reached the stomach the funnel should be raised as high as possible, to allow the escape of gases almost invariably present. It should then be lowered, in order to siphon out the fluid contents. If nothing escapes, the funnel is then to be raised and from two to six ounces of water poured into it from a pitcher; the funnel is then lowered and the water siphoned out. This procedure is repeated from four to ten times, or until the fluid comes back clear. About a quart of water is ordinarily used. Various solutions have been advised for stomach-washing, but nothing is better than boiled water, used at the temperature of from 100° to 110° F.—the higher temperature being employed when the gastric irritation is very great. If much tenacious mucus is present in the stomach an alkaline solution (bicarbonate of soda, 5j to 0j) is preferable. Through the tube are easily discharged mucus and small curds; larger ones are gradually broken down by repeated washing. Vomiting may be induced by over-distending the stomach with water. If there is great thirst there is often an advantage in leaving one or two ounces of water in the stomach. To this water it is at times beneficial to add lime water.

Stomach-washing in its application is practically limited to children under two and a half years. It is easiest in those under eighteen months.

Children of three years and over are usually so much alarmed and struggle so violently as to make it difficult and undesirable.

The indications for stomach-washing are: (1) Acute gastric indigestion, either with or without persistent vomiting. Here the purpose is



FIG. 13.—POSITION FOR STOMACH-WASHING.

simply to clear the stomach of its irritating contents, and a single washing may be sufficient. (2) Chronic indigestion attended by the production of gastric mucus. (3) Dilatation of the stomach. (4) Hypertrophic stenosis of the pylorus. (5) Poisoning.

**Gavage.**—Gavage consists in the introduction of food into the stomach by a tube passed through the mouth. The same apparatus is employed as in stomach-washing, and the method is similar, with the exception that for gavage the child should be placed upon the back, the head being steadied by an assistant. With older children a mouth-gag

is often necessary. After the tube has entered the stomach the funnel should be raised to allow the gas to escape. The food is then poured into the funnel; as soon as it has disappeared the tube is tightly pinched and quickly withdrawn, to prevent food from trickling into the pharynx, since this is often a cause of vomiting. If the food is regurgitated this usually happens at once. It may then be introduced a second time. After feeding, the child should be kept absolutely quiet upon the back.

In cases where all the food is given by gavage the interval between feedings must be considerably longer than under other circumstances. Often the food given should be partially predigested, since digestion in these cases is usually feeble. The stomach should be washed before each feeding, in order to remove mucus and to be sure that it is empty before the meal is given.

Gavage is valuable in the feeding of premature infants and after certain operations upon the mouth and neck. It is also useful, first, in the case of very young infants, who, suffering from severe malnutrition, can not be induced to take food enough to sustain life; secondly, in many acute diseases, particularly in septic cases where the child will not readily take the necessary food, as in diphtheria, scarlet fever, typhoid, pneumonia, etc.; thirdly, in many cases of cerebral disease where food is refused on account of delirium or coma; and, fourthly, in some cases of persistent vomiting, as first suggested by Kerley.

Gavage is a very simple procedure and one which a nurse can easily be taught. Not only may food be given, but also medicines and stimulants as may be required, with little or no trouble. The advantage of gavage over the continued coaxing or holding the nose and forcing the patient to swallow, will be at once apparent to one using it.

**Nasal Feeding.**—The method is similar to gavage, the only difference being that the tube is passed through the nose, and consequently a much smaller one is used. No. 10 American or No. 16 French scale is a proper size. Nasal feeding is applicable to children over two years old, in whom the tube can seldom be passed through the mouth without the use of a gag, and then only after much struggling. It is of value after intubation, tracheotomy, and other operations about the throat, also in some cases of throat paralysis, especially after diphtheria.

**Irrigation of the Colon.**—By irrigation of the colon is meant the flushing of the entire large intestine by fluids injected high up through a catheter or rectal tube.

The apparatus required for irrigating the colon is a fountain syringe, five or six feet of rubber tubing, and a flexible rectal tube or soft-rubber catheter—No. 26 or 27, French scale, being preferred. Kemp's double-current tube of hard or flexible rubber is useful. The same result can be obtained by using two catheters, the larger for outflow, the smaller for inflow. The child is placed upon the back, with the thighs flexed

and the buttocks brought to the edge of the bed or table. He should lie upon a Kelly pad or a rubber sheet so arranged as to form a trough emptying into a large basin or tub. The bag containing the water is hung two or three feet above the bed. If a catheter is used it is inserted just within the sphincter before the water is turned on. As it flows



FIG. 14.—COLON OF A CHILD SIX MONTHS OLD, IN POSITION. (From a photograph.)

the catheter is gradually pushed upward to a distance of twelve or fourteen inches. The water distending the intestine in advance of the catheter usually makes its introduction quite easy. In Fig. 14 is shown the colon of an infant of six months in position. It is the peculiar curve and the great length of the sigmoid flexure that make the introduction of water difficult, unless the tube is inserted for some distance.

Usually a pint, and often a quart, will be introduced before any water returns. At least a gallon of water should be used for a single irrigation. The washing should be continued until the water returns quite clean. Change of posture and gentle kneading of the abdomen should be employed during the irrigation, particularly the early part of it, to facilitate the introduction of the water into the upper part of the colon. At the end of the irrigation the rubber tube is detached and the water al-

lowed to escape through the catheter, which remains *in situ*. Sometimes as much as a pint of water remains in the intestine. This is usually passed within half an hour. As the irrigation of the colon almost invariably excites active peristalsis of the lower ileum, this part of the intestine is emptied as well. It is to be remembered that the colon of an infant six months old will hold about one pint without distention, and at the age of two years from two to three pints.

Irrigation of the colon is useful to clear this part of the intestine of mucus, faecal matter, undigested food, and the products of decomposition. It may also be employed as a means of local medication in ileocolitis. Where the object is simply to cleanse the intestine, a saline solution—a teaspoonful of common salt to a pint of water—is preferred.

The temperature of the water used for irrigation may be varied according to the special indications. For ordinary purposes, where cleansing only is aimed at, a temperature of from 95° to 100° F. seems to be best. When the body temperature is high, or when there is much pain, tenesmus and straining, cold water has important advantages.

Irrigation under most circumstances is required only once in twenty-four hours. It is important to use a large quantity of water. It must be done thoroughly to be of value, and either by the physician himself or an experienced nurse.

In collapse or great prostration hot saline injections may be employed for purposes of stimulation; the temperature of these should be from 105° to 110° F.

**Enemata.**—Simple enemata are useful in infants and older children for constipation. Where an immediate effect is desired the most efficient is one containing glycerine—e. g., for an infant, one teaspoonful to one ounce of water. Oil enemata (one half to one ounce) are useful where the faecal mass is hard and dry and expelled with difficulty. Enemata should always be given with care, and preferably a rubber catheter should be attached to the nozzle of the syringe.

Nutrient enemata have a limited application in infancy, as the rectum soon becomes intolerant. The quantity injected should be small, rarely more than one or two ounces, and the interval between injections should be at least four hours. In older children they may be used as in adults. For this purpose either completely peptonised milk or glucose may be employed.

The administration of drugs *per rectum* is useful in certain cases where, on account of the unpleasant taste or vomiting, the administration by mouth is difficult—e. g., quinine and chloral. As a diluent, gruel is preferable to water. If quinine is used, the bisulphate is the best preparation, but this must be well diluted. The temperature of enemata which are to be retained should be about 100° F. It is necessary in infancy to



press the buttocks together for half an hour afterward to prevent the expulsion of the injection.

**Hypodermic Medication.**—This is not so often used in young children as it should be, and is of the greatest service even in infancy. The use of morphine hypodermically in convulsions, of morphine and atropine in cholera infantum, of strychnine, adrenalin, caffeine, or digitalis in heart failure, may be cited as examples.

**Hypodermoclysis.**—This is a therapeutic measure of considerable value in a few conditions, chiefly when the system is suffering from a rapid loss of fluid as in some forms of acute diarrhœa, less frequently after severe hæmorrhage from whatever cause. A sterile normal salt solution is employed at body temperature and may be injected into any of the areolar planes of the body. With young infants the preferable location is between the scapulæ; next, the abdomen or the lateral thoracic region. For very small patients injections should be made at several points. The amount injected at one time may vary from one to four ounces for an infant, and from four to sixteen ounces for an older child. The apparatus should be carefully sterilised. One may employ a piston syringe with a connecting rubber tube and a hypodermic or fine exploring needle, or a funnel may be substituted for the syringe.

**Massage.**—In older children massage is useful for the same conditions as those for which it is employed in adults; the most important are anæmia, general malnutrition, chorea, and chronic constipation. It is necessary that in the beginning only the mildest movements of massage should be employed, and these but for a short time.

In infancy massage has a limited application, and it is doubtful whether it really does more than can be accomplished by the general friction of the body. This rubbing, either with the bare hand or with cocoa butter, or with some form of fat, is useful in malnutrition, in rickets, and in wasting diseases where the circulation is feeble and the muscular tone low. Cocoa butter is cleanly and has a pleasant odour, and is, I think, quite as valuable as the more commonly employed cod-liver oil, which is exceedingly disagreeable. The inunctions should be given daily after the morning bath, before an open fire. The rubbing should be continued for fifteen to twenty minutes.

**Anæsthetics.**—As a general anæsthetic for routine use, ether is to be recommended for children. Its disadvantages can largely be overcome by proper administration; in point of safety it is immeasurably superior to chloroform for the very young. The administration of ether to young children may be advantageously preceded by a few whiffs of nitrous oxide or ethyl chloride; both, however, are to be used with caution in infants. Ether should be given slowly, well diluted with air, and if used in this way its unpleasant features may be obviated. This can best be accomplished by the use of some special form of inhaler. Ether should

not be selected as the anæsthetic for patients suffering from nephritis, bronchitis, pneumonia, pleurisy, or any other disease attended by obstructed respiration. For all these conditions chloroform is much safer.

The dangers from chloroform are greatest when it is given too rapidly or in too concentrated a form. Both are exceedingly likely to occur where it is administered to a struggling child. The greatest care and judgment should be exercised at such times, or disastrous consequences may follow. To produce and maintain the effect desired with the minimum amount of chloroform should always be the aim. All anæsthetics, but especially chloroform, are dangerous in children with the so-called lymphatic diathesis. For the removal of tonsils or adenoids, chloroform should not be employed.

Nitrous oxide, while very useful in older children, as in adults, for momentary operations, is not well borne by infants. It produces so early and so deep asphyxia that its prolonged use may be fraught with serious danger.

## **PART II.**

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### **SECTION I.**

#### **DISEASES OF THE NEWLY BORN.**

##### **CHAPTER I.**

###### **ASPHYXIA.**

THE lungs in the full-term fœtus are of uniform dark red colour, and show very distinctly upon their surface the lobular divisions. They are firm and solid and readily sink in water. The connective tissue is very abundant, and forms distinct fibrous septa, which stretch through the lungs in every direction.

Inflation of the lungs begins with the first cry uttered by the infant as it is born into the world. The parts first expanded are the anterior borders of the lungs, then the upper lobes, and finally the lower lobes posteriorly. The superficial lobules are nearly always expanded before those in the interior of the lung. The inflation is sometimes irregular, because of the accumulation of mucus in some of the bronchial tubes. The right lung is frequently stated to be expanded earlier than the left. Although this is often the case, there is no uniformity in this respect. The important point to be remembered is, that the parts last inflated are the posterior portions of the lower lobes. The expansion of the lungs is a gradual process, and in healthy infants it is probably not complete much before the end of the second day. In delicate children it may be postponed for several days, or even weeks. The above statements are based upon post-mortem observations upon infants dying from various causes during the first weeks. It has often been a matter of great surprise to find at autopsy on an infant two or three days old, that less than one-half of the lung tissue was expanded, although the child had breathed well and shown no signs of atelectasis. Under normal conditions at full term inflation of the lungs takes place very readily, but not so readily in premature or delicate infants, on account of the feebleness of the respiratory muscles. The longer it is postponed after birth the more difficult does it become, on account of the changes which occur in the collapsed air vesicles. The condition of the child *in utero* may be described as one of fœtal apnœa, its oxygen being received and its

carbon dioxide discharged through the placenta, which is essentially the organ of respiration at this period. This condition is interrupted by cutting off the supply of oxygen and the accumulation of carbon dioxide in the blood. Which of these is the important factor in inducing pulmonary respiration has been much debated; but the best experimental evidence seems to show that it is the want of oxygen which stimulates the respiratory centres.

Under the term "asphyxia" may be included all cases in which primary respiration is not spontaneously established with sufficient force to maintain life. Usually there is no attempt at pulmonary respiration until after the birth of the child, but it may occur *in utero* or at any stage of parturition. Asphyxia may be of intra-uterine or extra-uterine origin.

**Etiology.**—1. *Intra-uterine Asphyxia.*—The maternal causes include any disturbance of the placental circulation during labour—anything which prolongs the second stage of labour, convulsions, hæmorrhage, the use of ergot in the second stage, or, finally, the death of the mother. The causes relating to the child are pressure upon the cord, multiple winding of the cord about the neck, early separation of the placenta, and pressure upon the brain. If the respiratory stimulus comes before the birth of the child, the effort at respiration may cause the entrance into the mouth and air passages of amniotic fluid, mucus, blood, meconium, etc.

2. *Extra-uterine Asphyxia.*—This condition is a much less common one. It arises from causes quite apart from those above mentioned, and depends upon malformations or intra-uterine disease of the organs of respiration, circulation, or of the brain. It may be secondary to an injury of any of these organs received during parturition. It is also seen in premature infants, where it depends upon the feeble development of the nerve centres and respiratory muscles and upon the soft, yielding chest walls.

**Lesions.**—In infants dying of intra-uterine asphyxia there are seen the usual changes found in death from suffocation, together with the effects of attempts at breathing *in utero*. There is general congestion of all the viscera, particularly of the brain and its meninges, the liver, and the lungs. They may show small, punctate hæmorrhages, and occasionally large extravasations. Blood or bloody serum may be found in any of the serous cavities. The right heart is overdistended with dark, soft clots, and the blood generally is more fluid than normal. The lungs may contain no air, but more frequently there are small, scattered areas in which lobular inflation has taken place. If the child has lived several hours there are larger areas of expanded lung, especially in the upper lobes, and these may even be emphysematous, if artificial inflation has been employed. In the mouth, nose, larynx, and even as far as the

finest bronchi, there may be found aspirated materials—amniotic fluid, blood, mucus, or meconium. In extra-uterine asphyxia there are organic changes in the viscera—malformations of the lungs or the heart, intra-uterine pneumonia or pleuritic effusion, malformation of the diaphragm and sometimes of the brain.

**Symptoms.**—Under normal conditions the newly-born infant begins at once to scream and to use his limbs, the purplish colour of the skin giving place in a few moments to a rosy pink. In the first degree of asphyxia—*asphyxia livida*—the child is deeply cyanosed. Either no attempt whatever is made at respiration, or it is superficial and repeated only at long intervals. The pulse is slow, full, and strong. The vessels of the cord are distended. Muscular tone is preserved, and also cutaneous irritability, so that with the application of almost any kind of external stimulus, respiration is excited and the symptoms disappear.

In the second degree—*asphyxia pallida*—the picture is quite a different one. The face is pale and deathlike, though the lips may still be blue. The heart's action is weak, and by palpation can rarely be felt at all. By auscultation the sounds are feeble, irregular, and usually slow. The cord is soft, pale, and flaccid, and its vessels nearly empty. The sphincters are relaxed, and meconium oozes from the anus. There is entire loss of tone in the voluntary muscles, so that the extremities and entire body seem perfectly limp. Cutaneous sensibility is abolished. The extremities are often cold. There may occur a few short, convulsive contractions of the respiratory muscles, but these are without effect and soon cease. Unless such cases receive the most prompt and efficient treatment, the heart's action becomes more and more feeble until it ceases and death occurs. Other cases are partly resuscitated and may survive for a few hours or days, when they gradually sink, respiration becoming more and more feeble in spite of all efforts to maintain it. Between these two extremes all degrees of severity are seen.

In extra-uterine asphyxia there may be some attempts at voluntary respiration continuing for several hours, sometimes for a day or two, but this may be inadequate to sustain life.

**Diagnosis.**—Almost the only condition with which asphyxia is likely to be confounded is cerebral compression from a meningeal hæmorrhage. The difficulties in the case are much increased by the fact that the two conditions are not infrequently associated. It may then be impossible to tell that in addition to asphyxia, intracranial hæmorrhage is present. If the hæmorrhage is extensive and the asphyxia only moderate, a diagnosis is possible in most of the cases. In hæmorrhage there is often a history of undue compression during delivery—sometimes the use of forceps. The fontanel is bulging; there is coma, and there may be paralysis. The respiratory murmur may be quite strong for several hours, but it gradually fails as the child becomes completely comatose.

Anæmia resulting from a large hæmorrhage, like that due to rupture of the cord, may simulate the severe form of asphyxia.

**Prognosis.**—This depends upon the grade of asphyxia and the treatment employed. There is but little tendency to spontaneous recovery in any form. In the milder cases recovery is almost invariable with any intelligent treatment. In the severest cases the outcome is always doubtful, although by persistent effort many infants that are apparently hopeless may be saved. In a prognosis as to the ultimate result, the frequent complication of asphyxia with meningeal hæmorrhage should always be kept in mind. Apart from this complication it is doubtful whether asphyxia has anything to do with the production of idiocy.

**Treatment.**—In every case the first step is to clear the mouth and pharynx of mucus by means of the finger covered with absorbent cotton. In the milder forms respiration is usually excited either by spanking the child or the alternate use of hot and cold baths. If the hot bath is employed, the water should be from  $104^{\circ}$  to  $108^{\circ}$  F. and always tested by a thermometer. After a moment the child should be dipped into very cold water, or the body may be douched with it. In the livid cases relief is often afforded by allowing the cord to bleed for a few moments before ligation. The loss of half an ounce of blood is ordinarily sufficient. Simply swinging the child in the air is a powerful stimulus to respiration. The above means will suffice in the great majority of cases. In the more severe forms, however, these are inadequate. There is no response whatever to external stimulation, either by heat or mechanical irritation. In these cases two methods of resuscitation may be employed: artificial respiration and direct inflation of the lungs.

One of the most widely employed methods of inducing artificial respiration is that of Schultze. The infant is grasped by both axillæ in such a way that the thumbs of the physician rest upon the anterior surface of the chest, the index fingers in the axillæ, and the remaining fingers extending across the back. The child is thus suspended at arm's length between the knees of the physician, the feet downward and the face anterior. The body is now swung forward and upward, until the physician's arms are nearly horizontal. This produces the inspiratory effort. When this point is reached, an arrest in the swinging causes flexion of the trunk, the head now being directed downward, the lower extremities fall toward the physician until the whole weight of the body rests upon the thumbs. In this way expiration is produced. Lusk cautions against the employment of this method if the heart's action is very feeble, as it may cause the heart to stop altogether. This method should be used with care and skill; clumsy and too forcible manipulation has resulted in many serious injuries to the viscera and fractures of ribs or clavicles.

A method introduced by Dew has been extensively employed in New York. The infant is grasped in such a way that the neck rests between


the thumb and forefinger of the left hand, the head being allowed to fall far backward, the upper portion of the back resting upon the palm of the hand; with the right hand the knees are grasped between the thumb and fingers, the thighs resting against the palm of the hand. Inspiration is produced by depressing the pelvis and lower extremities, thus causing the abdominal organs to drag upon the diaphragm, and at the same time gently bending the dorsal region of the spine backward. In expiration the movement is reversed, the head being brought forward and flexed upon the thorax, while at the same time the thighs are flexed so as to bring them against the abdomen. The body is thus alternately folded upon itself and unfolded as the movements are carried on. If there is much mucus in the mouth, the movement of expiration should first be made with the body completely inverted. This method is simple, efficient, and much less fatiguing than that of Schultze when it is to be maintained for a long time. It is also of great advantage in that it can be carried on while the child is in the hot bath, one of the greatest objections to the method of Schultze being the loss of animal heat incident to its use.

In all cases where artificial respiration is used the first movement should be that of expiration, to expel, so far as possible, mucus or other foreign substances from the air passages. The movements should be made from eight to twelve times a minute, and not too forcibly, the child being kept in the hot bath between the movements, and as much as possible during them. As long as the heart beats resuscitation is possible, and the case should not be abandoned.

Direct inflation of the lungs by the mouth-to-mouth method should not be employed.

An ingenious apparatus for artificial inflation of the lungs has been devised by Carrel of the Rockefeller Institute, making use of Meltzer's method of the continuous insufflation of air. A flexible catheter containing a wire stylet is introduced into the larynx. To the catheter is then attached the apparatus shown in Fig. 15. By means of the double bulb a continuous flow of air is maintained. The manometer shown at A measures the pressure employed and is a guide by which one is prevented from using an excessive amount of force. When the pressure employed is normal the mercury in the descending and ascending arms of the curved tube stands at about the same level; if an excessive amount of pressure is used, the mercury will be forced up into the bulb. Although this has been as yet very little employed in infants it has been extensively used in resuscitating animals and seems to fulfill all the indications better than any apparatus hitherto suggested. It is so simple of construction that it can easily be put together by any instrument maker.

The method introduced by Laborde, of making rhythmical traction upon the tongue ten or twelve times a minute as a means of exciting respiration, is sometimes very useful in conjunction with other methods.



Faradisation of the phrenic is of undoubted value, but somewhat difficult of application.

In cases of asphyxia it is not enough to make the child cry. The deep respirations should be made to continue, for very often it happens



FIG. 15.—CARREL'S APPARATUS FOR INFLATING THE LUNGS.

that resuscitation is only partial, and that the child after six or eight hours lapses into its previous condition. All severe cases require close watching for the first twenty-four or thirty-six hours, as a repetition of the treatment is often necessary.

## CHAPTER II.

### CONGENITAL ATELECTASIS.

THIS condition is one in which there is a persistence of the fœtal state in the whole or in any part of the lung.

Atelectasis is the pathological condition with which asphyxia of the newly born is usually associated. In most of the cases the condition of atelectasis is completely overcome by the means employed in resuscitation; in some, however, these means are only partially successful, so that a portion of lung of variable extent remains in the fœtal condition. These are the circumstances in which most of the cases of atelectasis arise. But there are others in which there is no history of early asphyxia, where the primary respirations, although taking place spontaneously, have not been of sufficient force and depth to produce full pulmonary expansion. This usually occurs in feeble infants, or in those who are premature. The causes of congenital atelectasis are therefore, in the main, those mentioned as producing asphyxia.

**Lesions.**—In cases where the child dies during the first few days the amount of expanded lung is often small, frequently not more than one



fourth of the pulmonary area. The expanded portion is usually the anterior borders of the upper lobes. This is often the seat of acute emphysema. The rest of the lung is still in the foetal state; it is of a brownish-red colour, very vascular, does not crepitate, and shows the lobular outlines both on the surface and on section. With a little force the atelectatic lung may be completely inflated.

If children have lived a longer time, nearly the whole of the upper lobes and the anterior portion of the lower lobes are usually well inflated. These portions are either normal or slightly emphysematous. The posterior portion of the upper lobes and the lower lobes are almost invariably the seat of the atelectasis. On the surface even these portions may present quite a large area of expanded vesicles, but the underlying portion may be solid to the touch, and crepitates but slightly. On section it is seen that only the most superficial part of the lung is inflated, while the interior of the lobe is unexpanded. Small hæmorrhages are frequently seen beneath the pleura.

It is usual for both lungs to be affected, and often, but by no means uniformly, to about the same degree. It is frequently a great surprise to discover that a child has lived for some weeks without presenting any signs of cyanosis, although using not more than one-third of its pulmonary area. This variety of atelectasis closely resembles the hypostatic pneumonia of delicate infants, and very often the two conditions are associated. It may require the microscope to decide between them. If congenital atelectasis has existed for a considerable time, there are usually found evidences of pneumonia. Inflation is not so easy as in recent cases, but with force the greater part of the lung can usually be expanded. The heart commonly shows the right auricle and ventricle to be distended with dark clots, and there is occasionally found a patent foramen ovale or some other form of congenital lesion. The liver and spleen are in most cases congested, and the spleen may be considerably enlarged. The mucous membrane of the stomach and intestines is sometimes deeply congested.

**Symptoms.**—In one group of cases the children are asphyxiated at birth, but the attempts at resuscitation have been only partially successful. Although the patients may live for a few days, there is cyanosis, which gradually deepens, and death takes place from asphyxia, exhaustion, or convulsions.

In a second group of cases the infants have been asphyxiated at birth, and resuscitated perhaps with difficulty, but to all appearance completely. They do not thrive, however, remaining small and delicate, gaining very little or not at all in weight, and showing poor circulation, cold extremities, and occasionally subnormal temperature. It is characteristic of these cases that the cry is never loud, strong, and lusty. Some of them will not cry at all. Such children may live several weeks. There may develop at any time, often quite suddenly and without assignable cause, attacks

of cyanosis with prostration. Children may have several such attacks, which do not excite suspicion since they pass away spontaneously. In other cases the symptoms are so severe that they may result fatally in a few hours, death being frequently preceded by convulsions. If energetically treated the symptoms may pass away but, reappearing in a few hours, or again after a week or more, they gradually deepen in intensity until death occurs.

Two cases that came under my observation in the New York Infant Asylum illustrate this point. The infants were twins, ten weeks old and delicate. Suddenly at night one child was taken with convulsions, became deeply cyanosed, and died in two and a half hours. It had been suffering from a slight attack of indigestion for a week previous. The other twin had been apparently well on the previous day. Two hours after the death of the first child the second was taken with similar symptoms, dying in a few hours. At autopsy I found very extensive atelectasis involving the posterior part of the upper and the greater part of both lower lobes. The lesions were almost identical in the two cases. In both, the stomach was greatly distended with food and gas. I have repeatedly seen the effect of overdistention of the stomach in producing cyanosis in young children, and in this instance I believe it to have been the exciting cause of the final symptoms. It was subsequently learned that during the six weeks of observation the nurse had witnessed several slight attacks of cyanosis in one of the infants. It is of course possible that the atelectasis in these cases may have been in part at least acquired.

I have seen a number of cases, in which there was nothing whatever to attract attention to the lungs until the final attack of cyanosis occurred. In not all of these cases is there a history of asphyxia at birth. Some are only puny, delicate or premature, exhibiting during the early weeks of life all the signs of feeble vitality. The subsequent course is the same as in those in which there is early asphyxia. The duration of life in these cases depends chiefly upon the extent of the atelectasis.

It is not to be supposed that all cases of congenital atelectasis terminate fatally. Infants in whom there is every reason to believe that atelectasis exists, from the occasional attacks during the first few weeks of cyanosis, feeble cry, poor circulation, etc., may under favourable conditions with improved nutrition recover completely, even though no special treatment is directed to the lungs.

**Diagnosis.**—The physical signs are of much less value than the symptoms. It should be remembered that the principal seat of the disease is the lower lobes posteriorly. Percussion usually gives resonance over the entire chest, although this may be somewhat diminished posteriorly. There is not, however, so much change as one would expect to find, for the collapsed areas are surrounded by others which are overdistended, and there are in the midst of the collapsed parts, especially upon the surface,

lobules which are inflated. If the two sides are involved to about the same degree, as is often the case, we can get no difference in the percussion note over the two lungs, and the change from the normal may be so slight as not to be appreciable. Where only one lung is affected a difference can usually be made out. The respiratory murmur is rarely bronchial, but generally only feeble in its intensity, and rather ruder in quality than normal. The cardiac sounds may be transmitted with abnormal intensity. As in the case of percussion, if only one lung is affected this is of some value in diagnosis, but it is not sufficiently marked to be readily recognised when both sides are involved. Occasionally râles are present.

**Treatment.**—In the newly-born child, whether asphyxiated or not, the physician should see to it that the infant not only cries, but does so loudly and strongly, and that this cry is repeated every day. If children do not cry naturally they must be made to do so by the alternate use of the hot and cold bath, as in cases of asphyxia, or by mechanical means, like spanking. This should be repeated at least twice a day, and continued for from fifteen to thirty minutes. It may seem cruel, but it is often the only means of saving life. Expansion of the lungs is much more easily induced during the first few days of life, becoming more and more difficult the longer it is delayed. Provided the condition is recognised, treatment is fairly successful. In institutions where delicate infants spend most of the time in their cribs, atelectasis is likely to be found. An infant needs exercise, and this is often only to be obtained by taking the child from its crib several times a day, by general friction, massage, the stimulus of fresh air, etc. Nothing is more certain to perpetuate atelectasis than to allow the infant a life of feeble vegetative existence. Food and feeding must be carefully attended to, but even these are of less importance than the maintenance of the animal heat. The temperature is often subnormal, and should be closely watched. If there is difficulty in keeping the child warm it should be rolled in cotton and surrounded by hot bottles, or kept in an incubator during the first few weeks. During attacks of cyanosis the same means are to be employed as in cases of asphyxia of the newly born—cutaneous stimulation and artificial respiration—the administration of drugs being of little or no value, but oxygen may be of assistance.

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### CHAPTER III.

#### *ICTERUS.*

SEVERAL varieties of icterus are met with in the newly born.

1. It is often seen in the various forms of pyogenic infection. In such cases the icterus is usually mild.

2. It may be due to congenital malformations of the bile-ducts.
3. It may depend upon interstitial hepatitis.
4. The most frequent of all varieties is the so-called idiopathic icterus, sometimes spoken of as "physiological" icterus.

In the cases included under the first and second heads icterus is a minor symptom. The other varieties are sufficiently important to require separate consideration.

**Malformations of the Bile-ducts.**—The common bile-duct is the most frequently affected. There may be atresia at the point where it opens into the intestine, the duct may be represented by a fibrous cord, or it may be absent altogether. In many cases this is the only lesion; in others it is associated with an impervious hepatic or cystic duct; in still others the common duct is normal, but the cystic or hepatic ducts are impervious.

At autopsy all the organs are usually found intensely jaundiced, particularly the liver. In recent cases this is very much swollen, but presents no marked organic changes. In cases which have lasted several months there is commonly found chronic interstitial hepatitis, sometimes to a very marked degree. This was present in nine of the fifty cases collected by Thomson. The gall-bladder is usually small, and often rudimentary. In cases of atresia of the common duct it may be greatly distended.

The condition of the bile-ducts is ascribed to an error in development and subsequent catarrhal inflammation. There does not seem to be sufficient evidence to prove that hereditary syphilis is an etiological factor of much importance. This was present in but five of Thomson's cases.

**Symptoms.**—The most striking symptom is jaundice, which is usually noticed a day or two after birth, and steadily increases until it becomes intense. The other symptoms of obstructive jaundice are present. The urine is coloured a dark brown or bronze by bile pigment, the stools are white, and tests show bile pigment to be absent, except in cases where malformation is limited to the cystic duct. The liver as a rule is much enlarged. The spleen is often swollen. Hæmorrhages beneath the skin or from any of the mucous membranes are quite common. Vomiting is usually absent. In most cases there is progressive wasting, and death from inanition within the first few weeks. Of Thomson's fifty cases, nine lived less than a month, and only eighteen over four months. Lotze has reported a case of a child living eight months with an impervious hepatic duct. A frequent cause of death in the more rapid cases is convulsions.

These malformations cannot be influenced by any treatment.

**Interstitial Hepatitis.**—There is seen in newly-born children a form of icterus which resembles the foregoing in many particulars, but which

may end in recovery. In three such cases which have terminated fatally I have found the lesions of a general interstitial hepatitis, presumably of syphilitic origin. It is not certain that syphilis is always the cause of this condition, for the clinical history in some of them gives no evidence of this disease. While not a common condition I believe it to be more frequent than congenital malformations of the bile-ducts with which it is often confounded.

The symptoms and course may be illustrated by the following cases: A full-term, well-developed child of eight pounds' weight became jaundiced on the second day. By the fifth day the jaundice was intense; stools, pale yellow, and urine deeply bile-stained. Examination at three weeks showed both liver and spleen much enlarged. The jaundice was very marked for over a month; it was nearly two months before it faded entirely. The nutrition of the child was a matter of much difficulty for several weeks. The enlargement of the spleen and liver like the jaundice disappeared very gradually. There was no other evidence of syphilis in this patient nor in the two other children of the family, and no history of this disease could be obtained in the parents. Yet the improvement which began with the use of mercurial inunctions strongly suggested a syphilitic lesion.

In another case, the symptoms and course of which were almost identical, the stools, though nearly white, never failed to give the reaction for bile. A previous child in this family had died three years before at the age of six weeks with persistent jaundice, which had been diagnosed congenital malformation of the bile-duct. There was no history of syphilis; but the mercurial inunctions seemed equally efficacious as in the first case cited.

Not much need be added to the symptoms described. Both in those recovering and in the fatal cases there was no fever and no ascites; but there was much tympanites. The application of the Wassermann test will no doubt aid in clearing up the etiology of these cases. Other evidences of syphilis should always be carefully sought, but in all the cases I have seen, even those ending fatally and with syphilitic lesions at autopsy, clinical evidence of syphilis during life was wanting. A careful trial of antisyphilitic treatment should, therefore, be made in every case of protracted jaundice in a newly-born child. One should not be too ready to make the diagnosis of malformation of the bile-ducts and regard the case as hopeless. Nor does the fact that the child recovers without antisyphilitic treatment exclude syphilis as the cause, for one of Still's cases recovered from the jaundice and died at the age of nineteen months, the autopsy showing lesions evidently syphilitic.

**Physiological or Idiopathic Icterus.**--In 900 consecutive births at the Sloane Maternity Hospital icterus was noted in 300 cases. In 88 it was intense, in 212 it was mild. According to the statistics of various lying-

in hospitals of Germany, it was found in from 40 to 80 per cent. of all infants. In the 300 cases just referred to, icterus was noticed on the first day in 4, on the second day in 19, on the third day in 72, on the fourth day in 86, on the fifth day in 67, and on or after the sixth day in 44. From the second to the fifth day is therefore the usual period for its appearance.

It usually increases in severity for one or two days and then slowly disappears. The average duration in the mild cases is three or four days; in those of moderate severity about a week; in the most severe cases it may last for two weeks. The icterus is first noticed in the skin of the face and chest, then in the conjunctivæ, then in the extremities. The skin varies in colour from a pale to an intense yellow. The urine in most cases is normal. It sometimes is of a light-brown colour, and only in the most severe cases does it contain bile pigment. According to Runge, both urea and uric acid are produced in larger amounts than in children not icteric. The stools are unchanged, the normal yellow evacuations occurring in the icteric as early as in those not affected.

According to some observers, in infants who are icteric the initial loss in weight is greater and the subsequent gain slower than in other children. This is not borne out by the Sloane statistics. Of the 300 icteric children, 155 made satisfactory progress in every respect and gained rapidly. The progress in 106 cases was said to be "fair"—i. e., at the time of discharge, usually on the tenth day, a slight gain in weight was noted. The remaining 39 did badly, not gaining in weight and showing other symptoms of malnutrition. The proportion of icteric infants who did well, moderately, and badly, was practically the same as of the other children in the institution not suffering from icterus. Icterus occurs with equal frequency in both sexes. According to Kehrer, it is more frequent in first children than in later ones, and considerably more frequent in premature children than in those born at term. The presentation, the duration of labour and its character—whether natural or artificial—have no influence upon the production of icterus. As a rule icteric children appear in other respects healthy, but in those below the average size the icterus is apt to be more intense.

Few subjects have given rise to wider speculation than this form of icterus. Its exact pathology is at present unknown. It is generally held that the icterus is due to resorption, and is hepatogenous in origin. The most recent and reasonable theory advanced is that of Abramow,<sup>1</sup> who considers it to be an anomaly of secretion of the liver cells; it is due to an active secretion of bile which occurs soon after birth and which is poured out into capillary ducts obstructed by thick bile which is present at birth; from these conditions there results an overflow of bile into

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<sup>1</sup> Knöpfelmacher, *Jahrbuch für Kinderheilkunde*, Vol. 17; 1908.

the lymph and blood vessels, producing jaundice. Usually the more feeble the child the more intense is the icterus.

In jaundiced infants who have died from accident or other causes the skin and almost all the internal organs are found icteric. There is also staining of the internal coat of the arteries, the endocardium, the pericardium, and the pericardial fluid. Sometimes the subcutaneous connective tissue is yellow, also the brain and cord; the spleen and kidneys only in the most severe cases. The liver is rarely discoloured. The bile-ducts are normal.

This jaundice is never fatal, and in itself is not serious. Other conditions, such as atelectasis, may co-exist, which may make the case grave.

**Diagnosis of the Different Varieties of Icterus.**—The diagnosis of physiological icterus is to be made from sepsis, malformations of the bile-ducts, and interstitial hepatitis. In sepsis the symptoms usually appear at a later date; there is fever, rapid wasting, and often a discharge from the umbilicus and local symptoms indicating peritonitis, arthritis, pneumonia, or meningitis. In malformations of the bile-ducts the icterus is usually more intense and appears almost immediately after birth; bile is absent from the stools; the icterus is persistent, and the symptoms go progressively from bad to worse, always ending fatally. In interstitial hepatitis the icterus develops at about the same time as, but is generally more marked than, in the physiological variety. Both liver and spleen are usually enlarged. The stools may be white, but still give a faint bile reaction.

Physiological icterus requires no treatment.

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## CHAPTER IV.

### *THE ACUTE INFECTIONS OF THE NEWLY BORN.*

It is possible for the newly-born infant to suffer from almost all of the common infectious diseases. Smallpox probably has been most frequently observed. In rare instances, influenza, typhoid fever, malaria, and pneumonia have occurred in the first days of life. As the mothers in many instances were suffering from the diseases during or just prior to delivery, the infants appear to have been infected before birth through the circulation of the mother. In other cases, especially in pneumonia, influenza, and gastro-enteritis, infection may take place soon after birth. The symptoms of these diseases in the newly born differ very little from those occurring in any other young infant. In addition to the diseases mentioned, there are other forms of infection which belong especially—some of them exclusively—to the newly born.

## THE ACUTE PYOGENIC DISEASES.

Under this head are grouped various infections of the newly born, due to the entrance of the common pyogenic bacteria. They have been designated as *puerperal fever of the child*, also as *pyæmia* or *septicæmia*, or simply as *sepsis of the newly born*. A variety of pathological and clinical conditions are met with. In some cases there is only a localised external inflammation, often terminating in abscess formation; sometimes one or more of the internal organs is affected; occasionally a general blood infection—a true septicæmia—is seen without any noteworthy local lesion; finally, there are the cases attended by the production of multiple abscesses in the viscera, joints, or cellular tissue—a true pyæmia. Formerly infections of this class were very common, especially in large lying-in hospitals; but, owing to the general adoption of the methods of aseptic midwifery, they have steadily diminished.

**Etiology.**—The source of infection of the child may be the vaginal secretion of the mother or, in rare cases, the mother's milk. Although it has been shown that in a great proportion of the cases the milk of a woman suffering from mastitis or from septicæmia contains pyogenic germs, still the taking of these into the stomach is not likely to infect the infant. More frequently the child is infected by the nurse in the process of dressing the cord, bathing, or cleansing the mouth or eyes, possibly after having attended to the needs of a septic mother or another child. Infection may be carried by the physician, by instruments, or by the dressings of the cord. Infection may occur through any wound or abrasion of the skin.

Infection through the umbilicus may take place either before or after the separation of the cord. The infection may take place through the umbilicus, yet this may give no external evidence of disease, although the umbilical vessels inside the body may contain pus. From this focus of infection may arise peritonitis, meningitis, or other inflammations. Entering through the mouth, bacteria may lead to infectious processes in the throat, the stomach or intestines, and rapidly produce death; or the alimentary tract may be the focus from which infection of distant parts may arise.

The micro-organisms chiefly concerned in these infections are the common pyogenic bacteria, *staphylococcus pyogenes aureus* and the *streptococcus*. The next in importance is the *gonococcus*, the rôle of which, especially in cases accompanied by joint suppuration, has only recently been appreciated. *Pneumococcus* infections occasionally complicate the others mentioned. While *streptococcus* infections are in general more serious than those due to the *staphylococcus*, some of the most severe ones met with belong to the latter class.



**Clinical Varieties.**—*Omphalitis.*—In this variety there is inflammation of the umbilicus, and cellulitis of the abdominal wall in the immediate neighbourhood. This results in the formation of an umbilical phlegmon. It may terminate in resolution, in abscess, or in gangrene. The usual termination is in abscess. These abscesses may be small and superficial, or they may be more deeply seated between the abdominal muscles and the peritonæum. Omphalitis usually begins in the second or third week of life, before the umbilicus has cicatrised. The process may result in erysipelatous inflammation and it may spread to the peritonæum.

*Inflammation of the Umbilical Vessels.*—This is one of the most frequent primary processes in pyæmic infection. The umbilical arteries are more frequently involved than the vein. According to Runge, inflammation of the vessels is always preceded by inflammation of the connective tissue which surrounds them, as the poison is taken up by the lymphatics and not by the blood-vessels. Omphalitis is frequently present, but in some cases the umbilicus shows nothing abnormal.

In arteritis the vessels may be involved to any degree: sometimes only a short distance from the abdominal wall, sometimes quite to the bladder. They contain pus, and often septic thrombi. Saccular dilatation is frequently present at several points. Pus sometimes exudes from the umbilical stump on pressure. The other lesions accompanying arteritis are those of pyæmic infection, more or less widely distributed. There are frequently peritonitis, suppuration of the joints, erysipelas, multiple abscesses of the cellular tissue, sometimes suppurative parotitis. Atelectasis is common. Pneumonia was found in twenty-two of Runge's fifty-five cases.

In cases of phlebitis, the umbilical vein is usually involved for its entire length from the abdominal wall to the liver. This may lead to an acute interstitial hepatitis going on to suppuration, or to phlebitis of the portal vein and some of its branches. In either case there is more or less parenchymatous hepatitis, and often multiple abscesses of the liver, most of the patients being jaundiced. Peritonitis also is a frequent complication.

*Peritonitis.*—This is one of the most frequent pathological processes in pyæmic infection, and is very often the cause of death. It is generally associated with umbilical arteritis, and often with erysipelas. In a considerable number of cases it is the most important lesion found. It may be localised or general. Localised peritonitis is generally in the neighbourhood of the umbilicus or of the liver. It may result in adhesions, or in the formation of peritoneal abscesses. More frequently the peritonitis is general, and resembles the septic peritonitis of adults. There is a great outpouring of fibrin coating the intestines and other viscera and the inner surface of the abdominal wall, causing adhesions

between the abdominal contents. Collections of sero-pus are found in the pelvis and in various pockets formed by the adhesions. Sometimes blood is present in the exudation.

The special symptoms which indicate peritonitis are vomiting, abdominal tenderness and distention, and protrusion of the umbilicus. The abdominal enlargement is chiefly from gas, but may be partly from fluid. There are present thoracic respiration, dorsal decubitus, flexion of the thighs and fixation of all the muscles, the child lying perfectly quiet. The temperature is usually but not necessarily high. Marked leucocytosis is generally present.

*Pneumonia*.—The most common form seen is pleuro-pneumonia. There is an abundant exudate of grayish-yellow fibrin covering the lung. Occasionally collections of pus are found in the sacs formed by the adhesions. Serous effusions are rare. The pulmonary lesion consists usually in a broncho-pneumonia, with consolidation of larger or smaller areas in the lungs—more often in the upper than in the lower lobes. It is not uncommon for minute abscesses to be found in the lung at various points. There is a purulent bronchitis of the larger and smaller tubes.

The symptoms are obscure and often indefinite. The only characteristic ones are cyanosis and rapid respiration, with recession of the chest walls on inspiration. The physical signs are inconstant and uncertain. Pneumonia often cannot be diagnosticated during life. In most of the fatal cases of pyogenic infection, whatever its type, there is found some involvement of the lungs. The changes are most extensive in cases in which the serous membranes are involved.

*Pericarditis* is rare and usually associated with pleurisy. Endocarditis is very rare. Hirst has, however, reported a case.

*Meningitis*.—When meningitis is present it is usually associated with peritonitis or with pleurisy. The lesions are those of acute purulent meningitis with a copious exudation, sometimes associated with meningeal hemorrhages, or with acute encephalitis and the production of multiple minute abscesses in the cortex. The local symptoms are often not marked, and are sometimes very obscure. The most characteristic are stupor, dilated pupils, opisthotonus, bulging fontanel, general rigidity, convulsions, and occasionally localised paralyses. The temperature is generally high. A positive diagnosis can generally be made by lumbar puncture, by which means also the exciting cause of the meningitis can usually be determined.

*Gastro-enteritis*.—Diarrhœa is a frequent symptom in all septic cases, constipation being rarely present. In many instances vomiting is a prominent symptom. In a small proportion of cases the most important local lesions are in the intestines, generally in the nature of a superficial catarrhal inflammation.

*Stomatitis*.—Infections of the oral mucous membranes are not infrequent but sometimes very severe. They may be due to the streptococcus, staphylococcus aureus or the gonococcus. An occasional complication of oral infections is abscess of the parotid.

*Osteomyelitis*.—Allard has reported a series of cases in which, after the general and local symptoms of pyogenic infection had existed for some time, suppuration occurred over various bones, especially the humerus, tibia, metatarsal bones, sacrum, etc. Trephining revealed the lesions of osteomyelitis. The abscesses usually made their appearance between the fourth and the sixth week. The most rapid case died on the fourteenth day, and none lasted more than two and a half months.

*Joint Suppuration*.—In certain pyæmic cases, and in some in which there are no other symptoms, acute suppuration in the joints occurs. This may come on very acutely in the first or second week, or more slowly as late as the second or third month. In the acute cases it is exceptional to have but one joint involved; frequently there are four or five. The small joints are rather oftener affected than the large ones, but almost any articulation in the body may be involved. With multiple joint suppuration there are present the general symptoms of pyæmia—high temperature, marked prostration, wasting, and usually secondary visceral inflammations develop. In those which occur late, or which develop more slowly, fewer joints are involved, often but a single one, the febrile symptoms are less marked or wanting. In my own experience, the organism most frequently found in these cases is the gonococcus; next to this in importance is the streptococcus and occasionally the pneumococcus is found. The joint lesion is usually a superficial one, the bones generally escaping. The gonococcus cases probably occur most frequently as a complication of ophthalmia; but I have seen several in which ophthalmia was not present and where the point of entry could not be determined.

Many of the abscesses supposed to be in the joints are shown at operation to be at the epiphyses: from this source the joints may be involved secondarily. A point to be remembered in the diagnosis of these joint inflammations is their resemblance to the epiphysitis of hereditary syphilis and other symptoms of that disease should be looked for. The confusion is increased by the fact that in syphilitic cases abscesses may follow as a consequence of a secondary infection.

*Abscesses in the Cellular Tissue*.—These are quite frequent, and may occur with suppuration in the joints or internal organs, or they may exist as the only lesion. They are nearly always multiple and may be found in almost any location. They vary in size from that of a small pea to one containing half an ounce of pus. They are due to the introduction of pyogenic germs, usually staphylococci. Their course is benign,

and they require no treatment except incision and cleanliness. When there is a disposition to their continued formation, the skin should be washed with an antiseptic solution.

**Erysipelas.**—This is seen especially during the first two weeks of life, and usually starts from the umbilicus or some abrasion of the skin, most frequently about the genitals or the scalp. When originating at the umbilicus it is generally complicated by other lesions, such as peritonitis and umbilical phlebitis. If it starts from any other part of the body it may be uncomplicated. Erysipelas beginning at the umbilicus gives rise to an area of induration and a circumscribed erythema. At first it may resemble a simple cellulitis; but the steadily increasing area of elevated induration and redness soon indicates the nature of the inflammation. From whatever point starting, the erysipelatous inflammation, owing to the feeble resistance of the tissues, in most cases spreads widely. The entire abdomen, chest, and back may be involved; and it may even spread to the extremities. Nearly the whole trunk may be affected in four or five days. It usually involves only the skin and superficial cellular tissue; but it may involve the deeper areolar planes and terminate in diffuse suppuration, or even in gangrene.

The constitutional symptoms are severe: great prostration, continuously high temperature— $102^{\circ}$  to  $105^{\circ}$  F.—rapid wasting, and frequently vomiting, diarrhoea, or convulsions are present. The disease is always serious, and usually fatal. It is often complicated by broncho-pneumonia. General œdema of the affected parts may persist for a few weeks after the inflammation subsides.

**Distribution of the Lesions.**—The frequency of the different visceral lesions in eighty-seven autopsies published by Bednar was as follows: Peritonitis in twenty-nine, pneumonia in fifteen, pleurisy in ten, meningitis in nine, meningeal hæmorrhage in eight, encephalitis in eight, cerebral hæmorrhage in four, entero-colitis in five, pericarditis in four. In thirty-one cases there was umbilical arteritis, and in nine cases umbilical phlebitis. There was one case each of pulmonary hæmorrhage, pleural hæmorrhage, acute hydrocephalus, acute bronchitis, and suppuration in the cellular tissue. Runge's later observations of thirty-six cases showed umbilical arteritis in thirty, umbilical phlebitis in three, and normal umbilicus in three. He found pneumonia in twenty-two of fifty-five cases. Other lesions frequently associated are atelectasis, swelling and softening of the spleen, cloudy swelling of the liver and kidneys, occasionally with foci of suppuration in these organs.

**General Symptoms.**—These may begin at any time during the first ten days—very rarely after the twelfth day. Fever is an exceedingly variable symptom—it may be very high; it may be almost absent; occasionally there is subnormal temperature. The course of the temperature is very irregular. Wasting is constant and quite rapid. It depends

upon the inability to take and digest food, upon the intestinal complications, and upon infection. In quite a number of cases wasting is almost the only symptom. Icterus is common; in many of the worst cases it is intense. It is met with where the liver is the seat of an acute parenchymatous or acute suppurative inflammation, and in many other cases where it depends apparently upon the blood changes. Hæmorrhages are common, and may be the direct cause of death. They may come from the umbilicus, the intestine, or almost any mucous membrane. They are sometimes subcutaneous, causing a general hæmorrhagic eruption. Nervous symptoms are generally present, and are sometimes marked. They are restlessness, rolling of the head, a constant whining cry, twitchings of the muscles of the extremities or face, stiffening of the body, more rarely general convulsions. Late in the disease, dulness and stupor are present. The pulse is rapid and weak and the respirations are often irregular, even when there is no cerebral complication. Diarrhœa is frequent; the stools are green, brown, sometimes black from the presence of blood, and are often very foul. Vomiting is less common. In addition to these there are symptoms due to the various forms of local inflammation—peritonitis, meningitis, pneumonia, erysipelas, subcutaneous suppuration and gangrene, these all being found in varying degrees and in various combinations.

**Prophylaxis.**—Pyogenic infection of the child, like puerperal fever in the mother, may be considered a preventable disease. Its occurrence is usually due to a failure to carry out proper rules regarding cleanliness and asepsis in connection with delivery. The statistics of the Moscow Lying-in Asylum, published by Miller in 1888, show that previous to the general introduction of aseptic methods, from six to eight per cent of all infants born in the institution died from some variety of infection. In twenty-three hundred successive labours at the Sloane Maternity Hospital, covering about eight years, not a single marked case occurred. From these figures it will be evident that in the vast majority of cases the occurrence of a case of infection of a serious nature is the fault of the physician or nurse in attendance.

The umbilicus should be cleansed and treated like any other fresh wound. Dry dressing should invariably be employed, and sterilised gauze or salicylated cotton in preference to household linen. If suppuration occurs at the time the cord separates, the parts should be cleansed daily with a bichloride solution, and a wet dressing of the same applied. The ligatures and everything which comes in contact with the umbilical wound should be sterilised. Careful attention should be given to the mouth, genitals, and all the muco-cutaneous surfaces, to prevent excoriations and intertrigo. Finally, every septic case occurring in an institution should be immediately isolated. A nurse in charge of a septic mother should not have the care of the infant.

**Prognosis.**—Pyogenic infections in the newly born, even in their mildest forms, are serious, and in their most severe forms almost always fatal. Very few cases recover in which erysipelas or any important visceral inflammation is present. The resistance of these patients is so feeble that the tendency of every inflammation is to spread, until they die from exhaustion. Only patients with localised inflammations, such as those of joints, skin, etc., are likely to get well.

**Treatment.**—This practically resolves itself into the treatment of individual symptoms as they arise. Wherever suppuration occurs, external abscesses should be evacuated and treated antiseptically. For the local inflammations of the lungs, peritonæum, and brain, little or nothing can be done in the way of direct treatment. Such inflammations are to be prevented, but can seldom be cured. The general indications are to look closely to the child's general nutrition by careful attention to all details of nursing and feeding, using stimulants whenever required by the condition of the pulse. For a local application in erysipelas, nothing in my experience has proven better than ichthyol ointment, ten to twenty-five per cent strength. It should be applied daily, spread upon muslin, which is then covered by gutta-percha tissue to prevent drying. In a disease so fatal as erysipelas, by ordinary treatment, vaccines should certainly be tried. In some cases they seem to have been of undoubted value.

#### OPHTHALMIA.

Ophthalmia of the newly born is to be classed among the pyogenic diseases. It usually consists in a purulent conjunctivitis. In the more severe cases there may be ulceration of the cornea, and even perforation into the anterior chamber of the eye.

The highly infectious nature of this ophthalmia is established. In the most severe cases the micro-organism generally found has been the gonococcus; but in the milder forms the gonococcus may be absent, and any of the common pyogenic germs may be found. In the gonococcus cases the infection occurs during labour, from the secretions of the mother, from the examining fingers of the physician, or from instruments; or after birth from infected cloths and other materials which come in contact with the eye. Healthy lochia produce only a catarrhal inflammation. The infection occurring after birth may take place at any time. That due to gonococcus infection from the mother is generally manifested on the third day, and is often virulent from the outset.

The symptoms are swelling of the lids, chemosis, copious purulent discharge, sometimes hamorrhages from the lids, ulceration, and there may even be sloughing of the cornea. The course of the disease depends upon the cause and upon the treatment employed. In the cases not

due to the gonococcus the course is generally benign, and with ordinary cleanliness usually results in recovery without any permanent damage to the sight. The gonococcus cases, unless energetically treated from the outset, are very frequently followed by permanent loss of vision. The best statistics upon the causes of blindness in adults show that from twenty-six to thirty per cent of such cases are due to ophthalmia in the newly born. This disease is occasionally complicated by other symptoms of gonococcus infection of a pyæmic nature. Many cases followed by acute articular symptoms have been observed.

Prophylaxis is of the utmost importance. Credé's statistics show that in 1874 the frequency of ophthalmia in his lying-in hospital was 13.6 per cent. In the three years ending 1883, among 1,160 newly-born children only one or two cases occurred. The method of prophylaxis which he adopted consists in dropping into the eyes of every child, immediately after birth, one or two drops of a two-per-cent solution of nitrate of silver. The general adoption of Credé's method, or of some similar means of disinfection, has resulted in a very great diminution in the frequency of ophthalmia throughout the world. These prophylactic means should be obligatory in all institutions, and should be used in all cases in private practice wherever there is any possible suspicion of the existence of gonorrhœa. In all other cases the eyes should be carefully cleansed with a saturated solution of boric acid. The use before delivery of an antiseptic vaginal douche is theoretically indicated, but practically it has been found to be inadequate for the prevention of the disease.

**Treatment.**—Everything which comes in contact with the eyes should be carefully disinfected. All cloths, cotton, etc., used for cleansing should be immediately burned. The strictest antiseptic precautions should be insisted on to prevent the spread of the infection by nurses. In institutions containing infants, severe cases of ophthalmia should always be isolated. The most important thing is to keep the eyes clean. In severe cases they must be cleansed every twenty minutes, night and day. It may be done by irrigation, or by using an eye-dropper with a bulbous tip, inserted alternately at the inner and the outer angle of the eye, and the fluid injected with force sufficient to empty thoroughly the conjunctival sac. Either a saturated solution of boric acid, or a 1-5,000 solution of bichloride, may be used in this way. Once or twice in twenty-four hours two or three drops of a ten-per-cent solution of argyrol should be used in each eye after cleansing with sterile water. Next to these measures is the use of cold. It may be applied as ice compresses which are changed every minute or two from a block of ice to the eye. These may be continued one-fourth of the time in the milder cases; in the severe ones almost constantly. When the cornea is involved the pupil should be dilated by atropine. If only one eye is affected the

sound one should be protected by covering it with a compress kept wet with an antiseptic solution.

## TETANUS.

Tetanus is an acute infectious disease characterised by tonic muscular spasm, which increases in severity by paroxysms occurring at longer or shorter intervals. It may be limited to the muscles of the jaw (trismus), or may affect all the muscles of the trunk, extremities, and neck.

The germ of tetanus usually gains access to the body of the infant through the umbilical wound. It exists in the soil, and the disease prevails endemically in certain localities. It is common in certain parts of Long Island and New Jersey. Among the negroes in some parts of the South it has for many years occurred with great frequency. It is stated that on one of the islands of the Hebrides every fourth or fifth child dies of tetanus. In a single house in Copenhagen eighteen cases were observed. Tetanus presents no essential lesions. It is rare except where dirt and filth prevail; but these alone are not sufficient to produce the disease. It is rare in the tenements of New York.

**Symptoms.**—These, as a rule, begin on the fifth or sixth day, or at the time of the separation of the cord. The first symptoms may not appear until the tenth or twelfth day, but rarely later than this. Generally the first thing noticed is difficulty in nursing, which, on examination, is found to be due to rigidity of the jaws (trismus). Nursing may be impossible on this account. The muscles of the jaw feel hard, the lips pout, and all the muscles of the face seem firm. Soon a slight stiffening of the body occurs, the child straightening the back as it lies upon the lap and continuing rigid for a moment or two. In the interval it is at first completely relaxed. These paroxysms soon increase in frequency until they may come on every few minutes, being excited by any movement of the body. The relaxation is then only partial, and the neck and extremities and sometimes nearly the whole body may become rigid and stiff as a piece of wood. The arms are extended, the thumbs adducted, and the hands clenched. The thighs and legs are extended, and no motion is possible at the hip or knee. The jaws can be separated slightly or not at all. The firm contractions of the facial muscles give a peculiar expression to the features. There is a low, whining cry. Swallowing is difficult, sometimes impossible. The pulse is rapid and soon becomes weak. The temperature at first is normal, but in the most acute cases rises rapidly to 104° or even 106° F.; in the milder cases it does not go above 101° F.

Death is due to exhaustion, to fixation of the respiratory muscles, or to spasm of the larynx. In the less severe cases all the symptoms are milder, and there may be intervals in which the rigidity is scarcely noticeable, so that respiration and deglutition may be carried on for some



time. In cases which terminate in recovery the temperature is but slightly elevated. The tonic contractions gradually become less severe, and the paroxysms less frequent. The children usually suffer for several weeks from the general symptoms of malnutrition, which are proportionate to the severity of the attack. Of eighty-eight fatal cases which are reported by Stadtfeldt all but five died between the ages of six and ten days. The duration of the disease in the fatal cases is seldom more than forty-eight hours, often less than twenty-four hours; in those terminating in recovery, between one and three weeks.

**Prognosis.**—Few diseases of infancy are more fatal than tetanus. Where it prevails endemically it is regarded by the laity as so uniformly fatal that usually no physician is called. Scattered through medical literature are quite a large number of isolated cases in which recovery has occurred. At the present time the proportion of fatal cases is probably between ninety and ninety-five per cent. Sporadic cases more frequently recover than those occurring in districts where the disease is endemic. The later the development of the symptoms, the slower their course, and the lower the temperature the more likely is the case to recover.

**Prophylaxis.**—A proper understanding of the nature of the disease has brought with it the means of rational prevention. The first essential is obstetrical cleanliness, which must include scissors, hands, dressings, ligatures—in short, everything which comes in contact with the umbilical wound. In districts where tetanus is endemic, thorough aseptic treatment of the umbilicus should be insisted upon, both at the first dressing and later, particularly at the time of the separation of the cord.

**Treatment.**—All drugs whose physiological action is that of motor depressants of the spinal cord have a certain amount of value in tetanus. The most important ones are chloral and the bromides. Nearly all the reported cures have been by one of these drugs or a combination of them. The mistake usually made is in using too small doses. Enough to produce the physiological effects of the drug must be given. The initial dose should not be large, but it should be repeated until the full effects are obtained. Chloral, however, has been the drug most generally relied upon. An hourly dose of one or two grains is usually required. If no effect is visible in ten or twelve hours the dose may be further increased, as the patient is in much greater danger from the disease than he can possibly be from the drug. Chloral may be given by the mouth or by the rectum, but must always be well diluted. The single case of recovery which I have seen was one treated by the bromide of potassium. This infant took eight grains every two hours for three days, afterward smaller doses. The child must at all times be kept as quiet as possible, without unnecessary handling or bathing. If nursing or feeding by the mouth is impossible, because the jaws cannot be separated, the child may be fed by a tube passed through the nose. This is

greatly to be preferred to rectal alimentation. Drugs may be administered in the same way.

*The Antitoxine Treatment.*—This is of especial value in prophylaxis. To be efficient as a curative measure it must be used early, for after the disease has developed it is very doubtful whether much can be accomplished by its use; but as it is harmless, it should be employed.

#### EPIDEMIC HÆMOGLOBINURIA (*Winckel's Disease*).

The essential features of this disease are hæmoglobinuria with icterus and cyanosis, this combination giving the skin a deeply bronzed hue (*maladie bronzée*). It is a rare disease, but has generally occurred epidemically in institutions. It is usually fatal. It is, without doubt, infectious, but its cause has not been discovered. Although generally called by the name of Winckel, who in 1879 made a report upon an epidemic of twenty-three cases, the disease was quite well described by Charrin in 1873, with a report of fourteen cases, and observed by Bigelow, in Boston, in 1875. All the cases included in Winckel's report occurred in one institution, affecting one-fourth of the children born during the period.

There is cyanosis, with a more or less intense icterus of the skin and internal organs. The umbilical vessels are usually normal. The kidneys are swollen, show small hæmorrhages into their substance, and under the microscope the straight tubes are seen to be filled with crystals of hæmoglobin, but contain no blood-cells. The bladder frequently contains brownish, smoky urine. The spleen is swollen and filled with blood pigment, which is diffused throughout the cells of the pulp, and free in the blood-vessels. Punctate hæmorrhages are seen in most of the other viscera.

The symptoms usually begin from the fourth to the eighth day after birth, and are fulminating in character, seldom lasting more than two days. There are rapid pulse and respiration, general restlessness, prostration, cyanosis, and general icterus, which may be intense. The temperature is normal or slightly elevated. There is rapid asthenia, often terminating in coma or convulsions. The urine is passed frequently, in small quantities. It is of a smoky colour, and contains hæmoglobin in considerable quantity, renal epithelium, and sometimes granular casts and blood-cells, but does not contain bile pigment. Albumin is sometimes present, but not in large quantity.

Treatment is of little avail, since all severe cases die.

#### FATTY DEGENERATION OF THE NEWLY BORN (*Buhl's Disease*).

A disease has been described by the author whose name it bears, the essential nature and causation of which are unknown. It occurs as

isolated cases, and is characterised by inflammatory changes leading to fatty degeneration in the viscera, especially the heart, liver, and kidneys; it seldom lasts more than two weeks, and is almost invariably fatal. Many of the lesions are similar to the ordinary post-mortem changes, and when found they should not be interpreted as pathological unless the autopsy is made within twelve hours after death.

The clinical features of this disease, as described, resemble those of pyogenic infection; and since the observations were made before modern methods of bacteriological study, it is highly probable that Buhl's disease is merely a form of pyogenic infection in the newly born.

#### PEMPHIGUS NEONATORUM—BULLOUS IMPETIGO.

Pemphigus is a term which designates a lesion rather than a disease. By it is meant an eruption of bullæ occurring usually upon a red base, the contents being in most cases clear serum. A condition somewhat resembling pemphigus sometimes follows the use in the newly born of too hot baths. Again, bullæ are seen as one of the lesions of congenital syphilis; they are then usually present at birth or appear soon after. They are most frequently seen upon the palms and soles. Infants so affected are generally in wretched condition, and soon die.

The only condition to which the term pemphigus neonatorum should be applied is quite different from both the preceding, and it has nothing in common with the pemphigus of later life. A better name is bullous impetigo, for its identity with impetigo contagiosa seen in older patients is now generally admitted. The disease is infectious, somewhat contagious, and occasionally occurs in small epidemics in institutions. Its spread in communities has been traced to midwives. The only important difference between this disease and the common impetigo contagiosa seen in older children, is its severity and its association with visceral infections. Most patients with bullous impetigo are delicate, neglected, and living in dirty surroundings; but not all are. I have seen it in robust infants who had received fairly good care.

The greater number of cases studied thus far have shown the presence in the blebs of the staphylococcus pyogenes aureus; less frequently the streptococcus has been the cause. The staphylococcus aureus was found in several typical cases occurring in my own hospital service. In one of these which came to autopsy, a general staphylococcus septicæmia was present.

The clinical picture presented by pemphigus neonatorum is so striking that it can scarcely be mistaken. The symptoms begin in most cases between the fourth and tenth day of life. The bullæ first appearing are scattered and often not larger than one-fourth or one-half inch in diameter. They may be seen upon any part of the body, but are

especially frequent about the face, hands, and other exposed parts. They rupture or dry and form crusts without suppuration. The small bullæ may gradually increase in size or several may coalesce until they cover an area two or three inches in diameter. As the disease progresses, new bullæ may appear over almost any part of the body. The skin is at first slightly reddened, then an exudation of serum occurs beneath the epidermis which loosens and slides upon the true skin. After rupture of the large bullæ, the epidermis at the margin forms a thin filmy border or hangs in shreds easily detached. The base of the large vesicles is a moist bright-red surface. When many have formed, the appearance closely resembles that seen after an extensive burn.

The course of the local symptoms is at first slow; then the bullæ may spread with great rapidity and death occur in from twenty-four to forty-eight hours. In less severe cases the course is more prolonged, the blebs are smaller, and recovery may take place.

The constitutional symptoms are at first wanting, but increase with the number and extent of the bullæ. There may be a slight rise of temperature or it may be subnormal. There is progressive weakness and



FIG. 16.—PEMPHIGUS NEONATORUM. Symptoms began on 13th day; death on 16th day of asthenia; temperature subnormal. The dark areas in the picture are entirely denuded of epidermis; they were formed by the coalescence of large bullæ.

great depression, much like that following a burn, and death occurs from exhaustion or from some visceral inflammation such as pneumonia or meningitis.

It is important to distinguish pemphigus neonatorum from congenital syphilis. In syphilitic cases, the liver and spleen are usually markedly enlarged, and other characteristic changes may be present in the nails, mucous membranes, or elsewhere.

Treatment is of little avail in the most severe cases, when the bullæ cover a considerable part of the surface of the body. The bullæ should be opened and drained, and the surfaces dressed with gauze covered with a two-per-cent ointment of white precipitate. There is little danger of

mercurial poisoning. When dressings are changed the skin should be sponged with a bichloride solution, 1-5,000 strength, or a one-per-cent solution of ichthyol or permanganate of potash. On account of the contagious nature of the disease cases occurring in institutions should be isolated.

## CHAPTER V.

### HÆMORRHAGES.

HÆMORRHAGES are quite frequent during the first days of life, and are important not only from the fact that they are often the cause of death, but, when the brain is the seat, from their remote effects. There are several conditions in the newly born which predispose to bleeding—the extreme delicacy of the blood-vessels, and the great changes taking place in the blood itself and in the circulation in the transition from intra-uterine to extra-uterine life. Hæmorrhages may complicate many of the diseases of the early days of life, such as syphilis or sepsis, or they may exist alone.

The cases may be divided into two groups: (1) Traumatic or Accidental Hæmorrhages, which depend upon causes connected with delivery; (2) Spontaneous Hæmorrhages, or The Hæmorrhagic Disease of the Newly Born.

#### TRAUMATIC OR ACCIDENTAL HÆMORRHAGES.

These are mainly due to pressure in natural labour, or to means employed in artificial delivery, but some of them may possibly result from injuries received before birth. They are more frequent in large children, in difficult labours, and where from any cause the body of the child has been subjected to undue pressure.

**Hæmatoma of the Sterno-Mastoid.**—Hæmatoma of the sterno-mastoid muscle leads to the formation of a tumour in the belly of the muscle. It is a rather rare condition, usually noticed in the second or third week of life, and it disappears spontaneously, rarely causing any permanent deformity. The tumour varies from three quarters of an inch to one inch and a half in length, being about the size and shape of a pigeon's egg. It is movable, almost cartilaginous to the touch, and sometimes slightly tender. The situation of the tumour is usually about the centre of the muscle. There is no discoloration of the skin.

In about two-thirds of the cases it occurs after breech presentations. It is much more frequent upon the right than upon the left side. In twenty-seven cases collected by Hænoch the right side was involved in twenty-one and the left in only six cases. The explanation of this differ-

ence is to be found in the obstetrical position. Rarely, both sides may be involved. The head is usually slightly inclined toward the shoulder of the affected side and rotated toward the opposite side. The swelling slowly diminishes in size, and in most cases by the end of the third month has nearly or quite disappeared. Occasionally a slight torticollis remains for a longer time, but in the majority of cases the recovery is perfect. Hæmatoma of the sterno-mastoid is due to the twisting of the head during parturition. It is not an evidence of the employment of any improper force in delivery. The twisting of the head produces laceration of some of the blood-vessels of the muscle, and in some cases there is doubtless rupture of some of the fibres of the muscle itself. Following this there occurs a certain amount of inflammation of the muscle and its sheath. The tumour is due partly to blood-extravasation and partly to inflammatory products. In one or two recent cases in which the sheath of the muscle has been opened it has been found filled with blood.

The condition requires no treatment. Operative interference is positively contra-indicated.

**Cephalhæmatoma.**—This is a tumour containing blood, situated upon the head, usually over one parietal bone, and tending to spontaneous disappearance by absorption. The source of the blood is the rupture of the small vessels of the pericranium.

**Etiology.**—Cephalhæmatoma is sometimes due to a distinct traumatism like the application of forceps or to some other injury during labour. In the majority of cases, however, there is no evidence of such injury. Besides the conditions predisposing to all hæmorrhages, there is the increased pressure in the blood-vessels of the head during delivery, especially when labour is prolonged or difficult; there may be changes in the bone, such as an imperfect development of the external table; and, finally, there may be changes in the blood itself. Cephalhæmatoma is a comparatively rare condition, being present, according to the statistics of the Sloane Maternity Hospital, in 20 of 1,300 consecutive births, or 1.6 per cent. The condition is more common after first, or difficult labours, and in vertex presentations; occurring twice as often in males as in females, probably from the greater size of the head.

**Lesions.**—In the 20 Sloane cases, the situation was over the right parietal bone in 12; over the left in 2; over both parietals in 4; over the occipital in 2. The location of the tumour seems to have a very close relation to the position of the head in the pelvis. In 8 of the right-sided cases the head was in the left occipito-anterior position. Of the cases with occipital tumours, both were breech presentations. Of the 16 cases with a single tumour the labour was natural in 10, tedious in 4, and in 2 forceps were used. Of the 4 double cases, 2 were forceps deliveries.

In rare cases triple tumours are met with, one over each parietal and

one over the occipital bone (Fig. 17). The attachment of the periosteum along the sutures usually limits the tumour to the surface of one bone. It never extends across the sutures or over the fontanel. In cases where there



FIG. 17.—TRIPLE CEPHALHEMATOMA.  
Infant seven days old.

is a more definite injury, such as from forceps, the tumour may be present over any one of the cranial bones, but more frequently over the parietal. The seat of the hæmorrhage is between the periosteum and the cranium. The scalp shows punctate hæmorrhages and sometimes infiltration with blood. In recent cases the blood is fluid; later it is coagulated. The amount of extravasated blood is usually from half an ounce to an ounce. The cases following natural delivery are generally uncomplicated. The traumatic cases may be complicated by ex-

travasations between the bone and the dura (internal cephalhæmatoma), or by meningeal or cerebral hæmorrhages. If there is a wound, infection may be followed by purulent meningitis and even by cerebral abscess.

*Symptoms.*—The tumour is usually noticed from the first to the fourth day after birth, appearing as a slight prominence in one of the positions mentioned. Gradually increasing in size, it attains its maximum at the end of a week or ten days, and then slowly diminishes. In size and shape the usual tumour may be compared to the bowl of a tablespoon. In marked cases it may be one-third the size of the child's head. To the touch it is soft, elastic, fluctuating, and irreducible. It does not increase with the cry or cough. There is no extra heat and no signs of inflammation. Usually the tumour does not pulsate, although in rare instances pulsating cephalhæmatomata have been seen. Very soon the tumour is surrounded by a marginal ridge. At first this is apparently from coagulation of blood, but later it may be bony. The prominent ridge with the soft centre gives a sensation somewhat like that of a depressed fracture. Sometimes on pressure there is obtained a sort of parchment-crackling. This is generally found as the swelling is subsiding, and is sometimes clearly due to the formation of minute bony plates upon the inner surface of the periosteum. It may be found when there is nothing but thin coagula to explain it. In certain cases following severe traumatism, cephalhæmatoma may be complicated with wounds of the scalp, fracture of the skull, and even

lacerations of the dura mater or the brain. In such cases the tumour may become inflamed, but in the spontaneous cases this is extremely rare. The usual signs of abscess develop, which may open externally or burrow. Fortunately this termination is seldom seen.

As a rule, without any interference, the uncomplicated cases go on to recovery. The complete disappearance of the tumour may be expected in from six weeks to three months, depending on its size; but a hard, uneven elevation may remain at its site for a longer time. The cases due to severe traumatism are more serious, the gravity depending not upon the cephalhæmatoma but upon the complicating lesions.

*Diagnosis.*—Cephalhæmatoma may be confounded with encephalocele. This, however, occurs along the line of the sutures or at the fontanels, is partially reducible, pressure causes cerebral symptoms, and frequently the tumour increases with respiratory movements. Caput succedaneum often appears in the same place as a cephalhæmatoma and at the same time, but this is an œdematous, not a fluctuating tumour, and begins to disappear by the second or third day. From a depressed fracture of the skull, it is differentiated by the fact that in cephalhæmatoma there is a tumour and not a depression; the prominent margin which is raised above the contour of the skull, is not osseous and the skull can be felt at the bottom of the centre of the tumour.

*Treatment.*—The treatment in the uncomplicated cases is simply protective, all such cases tending to spontaneous recovery. No local or general treatment to promote absorption is required. The child should be so placed and so handled that no injury may be done to the affected part. Compresses are unnecessary. If complications exist, such as injury to the bones, dura, or brain, they are to be treated in accordance with general surgical principles. Operative interference is called for only when suppuration has occurred, or when there are brain symptoms which point to the existence of internal as well as external cephalhæmatoma.

*Visceral Hæmorrhages.*—While these are most frequent in large children and following difficult labours, they may occur in small children and where the labour has been easy and normal—their occurrence here being due to the feeble resistance of the blood-vessels. From one hundred and thirty autopsies upon still-born children or those dying soon after birth, Spencer concludes that intracranial hæmorrhages are more frequent in head-forceps than in breech cases, and more frequent in breech than in natural vertex deliveries. Other visceral hæmorrhages are much more frequent in breech cases.

Not all visceral hæmorrhages are to be classed as traumatic. They are often seen with the spontaneous hæmorrhages from the skin or mucous membranes. When, however, they are single, they seem to be of traumatic rather than of pathological origin.

The most important of the visceral hæmorrhages are intracranial.



These are discussed in the chapter devoted to Birth Paralyses. Rarely there may be large hæmorrhages into the lung. Here the blood fills the air vesicles and the small bronchi, and coagula may be found even in the larger bronchi. A large part of a lobe or an entire lobe may be involved. On section the condition resembles atelectasis, and it may give the physical signs of consolidation.

The abdominal viscera suffer more than those of the thorax because less protected against pressure. Small hæmorrhages are not uncommon upon the surface of any of the viscera covered by peritonæum. Intra-peritoneal hæmorrhages are rare, but may be very extensive, amounting to one or two pints. Sometimes no ruptured vessel can be found. The hæmorrhage may be primarily in the peritoneal cavity, or it may result from rupture of one of the viscera, especially the suprarenal capsule. It may be large enough to produce death from loss of blood.

Small surface hæmorrhages of the liver are not infrequent. Occasionally one of considerable size occurs separating the peritoneal covering and forming a tumour generally upon the superior surface. Such laceration may be produced during labour, and a slow accumulation of blood may take place beneath the capsule, death resulting from rupture into the peritoneal cavity. Laceration of the capsule of the liver in a still-born infant has been reported. Of the large hæmorrhages, those into the suprarenal capsules are perhaps the most frequent. The capsule may be distended to nearly the size of an orange, the kidney being surrounded by a mass of blood-clots. Blood may be extravasated into the retroperitoneal connective tissue, and rupture may take place into the peritoneal cavity.

Except in the intracranial variety, visceral hæmorrhages cause few symptoms, and in the great majority of cases the diagnosis is not made. Intrapulmonary hæmorrhages have given rise to the signs of consolidation of the lung and even to hæmoptysis. The abdominal hæmorrhages are the most obscure. There may be a general abdominal distention with the usual symptoms of loss of blood, or there may be a circumscribed swelling. In many cases nothing is noticed until a rupture of a subperitoneal hæmorrhage takes place into the general peritoneal cavity, when there may be sudden collapse and death.

The visceral hæmorrhages are not amenable to treatment. The prognosis depends upon the size and position of the hæmorrhage. In the cases of abdominal hæmorrhage the diagnosis is extremely obscure and is rarely made during life.

#### SPONTANEOUS HÆMORRHAGES—THE HÆMORRHAGIC DISEASE OF THE NEWLY BORN.

A disposition to bleeding is seen with many diseases of the first few days of life, especially those of an infectious character, like syphilis and

pyæmia. With most of these, however, the hæmorrhages are small, and the condition may be compared to the hæmorrhagic tendency seen in certain forms of infection of later life, such as measles, smallpox, and malignant endocarditis. There is, however, a class of cases in which the hæmorrhages are not associated with any other known process, and in which the escape of blood from the small blood-vessels is the chief or essential symptom. In these cases the bleeding is much more extensive than in the others mentioned. These hæmorrhages are characterised by the fact that they are spontaneous in origin, having no connection with delivery, they are multiple in location, and, while little influenced by treatment, they tend to cease spontaneously after quite a limited time. They are most often from the umbilicus, the mucous membranes of the stomach and intestines, or beneath the skin, but they may be from almost any mucous surface or into any organ of the body.

**Etiology.**—These hæmorrhages are not common, and are met with much more often in institutions than in private practice. In 5,225 births in the Boston Lying-in Asylum, Townsend reports 32 cases of hæmorrhage, or 0.6 per cent. In the Lying-in Asylum of Prague, Ritter observed 190 cases in 13,000 births, or 1.4 per cent. In the Foundling Asylum of Prague, Epstein reports hæmorrhages in 8 per cent of 740 infants.

The condition is not a manifestation of hæmophilia. Of 576 bleeders collected by Grandidier, only 12 had a history of hæmorrhage at the time of the falling off of the cord, and symptoms very rarely appeared before the end of the first year. Hæmorrhages in the newly born are only slightly more frequent in males, while in hæmophilia they predominate 13 to 1. The hæmorrhagic disease of the newly born is self-limited, and runs a definite course to recovery or death. The tendency to bleed does not extend beyond a few weeks, and often lasts but a few days. Circumcision has been done within a few days after the cessation of the hæmorrhages without any unusual bleeding. In a case under my observation with the most extensive subcutaneous hæmorrhages I have ever seen, all tendency to bleed had ceased before the separation of the cord, although there had previously been bleeding at the navel. The bleeding occurs with about equal frequency in feeble and in well-nourished infants. Syphilis is associated in but a small proportion of the cases. On the other hand of 132 cases of congenital syphilis observed by Mracek, only 14 per cent suffered from hæmorrhages.

A more frequent association with sepsis has been noted. Of the 61 cases observed by Epstein not less than 29, and of the 190 cases of Ritter, 24 were associated with sepsis. During one year there were 8 marked cases of hæmorrhage in the Nursery and Child's Hospital in about 225 deliveries. While more cases of sepsis occurred among the

children during that year than usual, it was striking that not one of these hæmorrhagic cases gave any evidence of sepsis, and that none of the septic cases had bleeding. An epidemic of 10 cases of hæmorrhages among 54 births at the New York Infirmary for Women and Children was studied in 1899 by Kilham and Mercelis. These all occurred in the course of two months; the epidemic ceased as soon as the cases were properly isolated.

The circumstances in which the hæmorrhagic disease occurs point strongly to an infectious origin. Quite a number of these cases have now been studied bacteriologically, but with no uniform results.

While these hæmorrhages are not traumatic, bleeding is exceedingly prone to occur in the skin over pressure points such as the back, the elbows, the occiput, and the sacrum. It is also common from the mucous membranes which are the seat of pathological processes, especially from the eyes, the nose, and the genitals.

**Lesions.**—In very many of the cases the autopsy shows nothing except the hæmorrhages in the various situations and the blanching of the organs due to the loss of blood. The hæmorrhages of the brain are usually meningeal and diffuse. They are considered more at length in the chapter upon Birth Paralyzes. The pulmonary hæmorrhages are usually small and unimportant, and large hæmorrhages into the pleura or pericardium are very rare. The stomach and intestines may contain considerable blood variously disorganised in the different parts of the canal, and there may be ecchymoses of the mucous membrane. In addition, ulcers may be found in the stomach and duodenum. In twenty-four autopsies upon cases with hæmorrhage from the stomach and intestines collected by Dusser, ulcers were found in the stomach in nine cases, and in the intestines in four. These ulcers are multiple, small, and usually superficial, but may extend to the muscular coat and may even perforate. The intestinal ulcers are found only in the duodenum and resemble those of the stomach. The cause of these ulcers is somewhat obscure; some of them are undoubtedly dependent upon inflammatory changes, probably of infectious origin; others have been compared to the peptic ulcers of later life, and are attributed to thrombi in the blood-vessels of the mucous membrane. These ulcers are found in but a small proportion of the cases in which bleeding occurs from the alimentary tract, and they may be wanting even where it has been very profuse.

Small extravasations may be seen upon the surface or in the substance of any of the abdominal organs. The changes found in the blood have not been uniform and have as yet been only imperfectly studied.

**Symptoms.**—The onset is most frequently in the first week of life; very rarely after the twelfth day. The hæmorrhages are usually multiple. Their location in Ritter's 190 cases was as follows: Umbilicus,

138 (umbilicus alone, 97); intestines, 39; mouth, 28; stomach, 20; conjunctivæ, 20; ears, 9. In Townsend's 50 cases: Intestines, 20; stomach, 14; mouth, 14; nose, 12; umbilicus, 18 (umbilicus alone, 3); subcutaneous ecchymoses, 21; abrasion of skin, 1; meninges, 4; cephal-hæmatoma, 3; abdomen, 2; pleura, lungs, and thymus, 1 each.

In many cases nothing is noticed until the hæmorrhage begins. The first bleeding noticed may be from the stomach, intestines, or any of the mucous surfaces, beneath the skin, or from the umbilicus. The amount of blood lost in most cases is not great, but there is a continuous oozing. The total hæmorrhage may be only a few drachms or it may reach several ounces. The general condition is one of considerable prostration, often from the outset. In all cases there is rapid loss of weight. The temperature may be high, low, or subnormal. A marked elevation of temperature may depend not upon the hæmorrhage but upon associated conditions. In a large number of the cases there is diarrhœa.

The duration of the disease in cases which recover is usually but one or two days. In fatal cases it is rarely more than three days, and often less than one. Death may result from the gradual failure of all the vital forces or from rapid loss of blood.

*Umbilical Hæmorrhage.*—A slight oozing from the umbilicus not infrequently occurs when the ligature has been improperly applied. This is generally controlled by simple measures. Spontaneous hæmorrhage is quite different. It is rather later than bleeding from the mucous membranes, usually occurring between the fourth and the seventh day. There may be bleeding into the cord as well as from its free extremity. A slight stain upon the dressing is usually the first note of warning, but in exceptional circumstances a gush of blood is the first symptom. The hæmorrhage may be temporarily arrested by various means, but it shows a strong tendency to recur in spite of everything which is done. The usual duration is two or three days. It has been known, however, to persist for twelve or fourteen days, and it may be fatal in less than twenty-four hours from the time it is noticed.

*Hæmorrhage from the Stomach and Intestines.*—Bleeding occurs much less frequently from the stomach than from the intestines. The latter is called *melæna*. Gastro-enteric hæmorrhages begin, in the great majority of cases, during the first three days of life. The blood vomited is usually in dark-brown masses, and not very abundant; more rarely it is bright red. The quantity varies from one drachm to half an ounce. Vomiting is liable to be excited by nursing. The blood discharged from the bowels is always dark coloured, usually intimately mixed with the stool, very rarely in clots. If in doubt between blood and meconium, one should look for the corpuscles with the microscope. When this is not conclusive on account of the disorganisation of the corpuscles, a chemical test for hæmoglobin should be made. Concealed hæmorrhage

into the stomach may take place, which may even be sufficient to produce death, no blood being vomited or passed by the bowels. In such cases the autopsy may reveal quite a large quantity of blood both in the stomach and intestines.

*Hæmorrhage from the Mouth.*—The quantity of blood is rarely large; but it is here that it is often first seen. Its source may be the mucous membrane of the mouth, pharynx, œsophagus, stomach, or bronchi. It may be associated with ulceration of the hard palate, with thrush, or with fissures of the lips.

*Hæmorrhages from the nose* are infrequent, and are more often due to syphilis than to other causes. These are rarely profuse, but are frequently repeated.

*Subcutaneous Hæmorrhages.*—These often appear in places exposed to pressure, such as the sacrum, heels, occiput, or back, but may occur anywhere. In some cases these hæmorrhages are very extensive, as in one recently under observation, where nearly one-third of the thorax was covered. Where they occur alone or form the principal lesion, the prognosis is favourable.

*Hæmaturia.*—The urine is not only stained with blood, but sometimes contains clots. This hæmorrhage may have its origin in the bladder, urethra, or kidney. Blood coming from the kidney is sometimes due to the irritation of uric-acid infarctions, and may have nothing to do with the general hæmorrhagic disease.

*Hæmorrhage from the Conjunctiva.*—The blood usually comes in drops from between the eyelids, chiefly from the tarsal surface. It is generally preceded by conjunctivitis.

*Hæmorrhage from the Female Genitals.*—This not infrequently occurs without hæmorrhages elsewhere, and under such circumstances is rarely serious. Cullingsworth collected thirty-two cases in children under six weeks of age—no case having resulted fatally. These are not to be regarded as cases of precocious menstruation.

**Diagnosis.**—This is generally easy, as the hæmorrhages are usually multiple and some of them external. A slight hæmorrhage from the intestine may be easily overlooked. Large hæmorrhages into the internal organs also are obscure and not often recognised. Spurious hæmorrhages from the stomach may occur, blood being vomited which has been swallowed during birth or nursing. The source of bleeding may also be the mouth, nose, or pharynx, and sometimes blood is swallowed in large quantities and afterward vomited. These cavities should therefore always be examined, since local treatment may be efficacious. Syphilis should be suspected when the bleeding is chiefly nasal.

**Prognosis.**—In all circumstances the hæmorrhagic disease in the newly born has a bad prognosis. Of 709 cases collected by Townsend, the mortality was 79 per cent. In any single case the prognosis depends

upon the extent and severity of the hæmorrhage, upon the vigour of the child, and upon how well it can be nourished. No case should be looked upon as hopeless, for perfect recovery has repeatedly taken place where it seemed impossible.

**Treatment.**—Local measures may be employed in all external hæmorrhages with some prospect of benefit. The bleeding points may be touched with persulphate of iron or with chromic acid fused upon a probe, or a solution of adrenalin chloride may be used. These measures may be employed alone or in combination with pressure.

Although recoveries have been reported following the use of a great variety of remedies, it is by no means established that the result was due to the drugs employed. Many of the milder cases recover without any special treatment. On the whole, the medicinal treatment is very unsatisfactory. The drug which has been most in favour is adrenalin, which can be used internally in the form of suprarenal extract. I have myself seen one case in which decided benefit apparently followed its use in severe gastric hæmorrhage. Two grains or more can be given every two hours. Gelatine has many advocates. It is used by subcutaneous injection. A two-per-cent solution which has been twice sterilised is employed, from 40 to 50 c.c. being administered two or three times daily. Calcium lactate in some instances appears to exert a positive effect. It may be given in frequently repeated doses up to 20 or 30 grains a day.

The latest addition to the treatment of this condition is the use subcutaneously of human blood serum. This was first suggested by J. E. Welch, of New York, who has tried it in seventeen cases. His results have been confirmed by others. Of the efficacy of this treatment there can be no longer any question. Whether all cases of hæmorrhage of the newly born are due to the same cause and therefore amenable to the same treatment is not yet established. Whenever possible, therefore, injections of blood serum should be tried for these patients. The serum may be obtained from the blood of any healthy adult under sterile conditions; the quantity used should be from 40 to 50 c.c. injected three times a day. Larger doses may be used without danger. It should be repeated as long as any tendency to hæmorrhage exists. Usually, however, if it acts at all it does so promptly. In most patients all bleeding ceases in twenty-four hours after the first injection.

The brilliant results which have followed transfusion as first practised by Carrel should lead to its use in the event of failure by other means, whenever it is practicable to adopt it, no matter how grave the symptoms may be. The general treatment should have reference to maintaining the nutrition by careful feeding, judicious stimulation, and attention to the circulation, the body temperature, and the general condition of the child.

## CHAPTER VI.

*BIRTH PARALYSES.*

BIRTH paralyses are chiefly due either to pressure upon the child by the parts of the mother or to artificial means employed in delivery. They may be cerebral, spinal, or peripheral.

*Cerebral paralyses* are in almost every instance due to meningeal hæmorrhage, and accompanied by a certain amount of injury to the brain substance. Very infrequently they depend upon cerebral hæmorrhage, laceration of the brain, or pressure from a depressed fracture.

*Spinal paralyses* are extremely rare, and only a few examples are on record. They are due to laceration of, or hæmorrhage into, the cord or its membranes. These lesions produce paraplegia, the exact distribution of which depends upon the point at which the cord is injured.

*Peripheral paralyses* usually affect the face or the upper extremity. Paralysis of the face is due in most cases to the application of forceps. Paralysis of the upper extremity is most frequently of the "upper-arm type," and is known as Erb's paralysis. It usually follows extraction in breech presentations. Peripheral paralysis of the lower extremity is almost unknown.

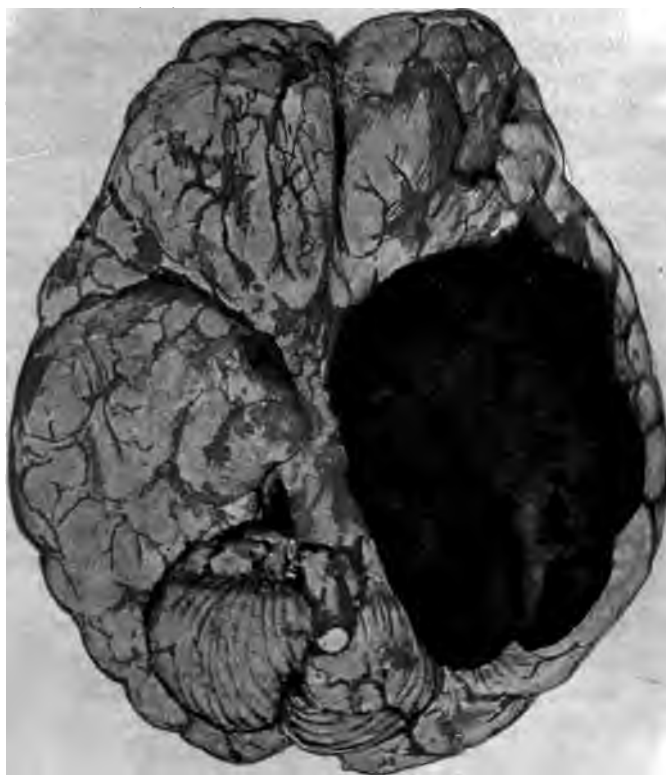
## CEREBRAL PARALYSIS.

Cerebral paralysis is often used synonymously with meningeal hæmorrhage. This lesion is not infrequent, and is of great importance not only from its immediate effects, but because upon it depend many of the cerebral paralyses seen in later life. According to Cruveilhier, at least one-third of the deaths of infants which occur during parturition are due to this cause.

**Etiology.**—The same predisposing causes exist in the cases of meningeal hæmorrhages as in others occurring at this time. A small number of cases are associated with syphilis; others with pyogenic infection. In a few cases there is a history of an injury—usually a fall or blow upon the abdomen—during the last months of pregnancy. Meningeal hæmorrhage may occur as one of the lesions in the hæmorrhagic disease of the newly born. The most important causes, however, are connected with parturition. These hæmorrhages are essentially mechanical, and are favoured by everything which increases or prolongs pressure upon the head. The conditions with which they are associated are tedious labour, breech presentations with difficulty in extracting the head, instrumental deliveries, and premature births. The majority occur in first-born children. In many of the cases there is also a hæmorrhage outside the skull.

**Lesions.**—These hæmorrhages are more common at the base than at the convexity, and at the posterior, than at the anterior part of the skull.

## PLATE II.



### MENINGEAL HÆMORRHAGE IN THE NEWLY BORN.

From a patient in the Nursery and Child's Hospital, dying on the sixth day. Primary respirations poor; child very dull and apathetic, refused to nurse; once vomited blood and had an ecchymosis of the right conjunctiva. On the last day, high temperature (105° F.) and general convulsions. Some changed blood found in the stomach and intestines at the autopsy; brain greatly congested, and at the base was the clot shown in the picture.





They are most frequently found over the cerebellum and the occipital lobes of the cerebrum. The entire extravasation is often beneath the tentorium. The extent of the hæmorrhage is exceedingly variable. There may be a single large clot at the convexity or at the base (Plate II), the hæmorrhage may be limited to the convexity of one hemisphere, or it may cover nearly the entire surface of the brain. Diffuse hæmorrhages are more common than a single circumscribed clot. In cases with vertex presentations the principal lesion is usually at the base, and often limited to that region. In breech cases it is more frequently at the convexity. The source of the blood may be a laceration of one of the sinuses of the dura mater caused by overlapping of the parietal bones. But more frequently the blood comes from one of the cerebral veins, or from the capillary vessels of the pia mater. In thirty-seven of Bednar's fifty-two cases, the extravasation was beneath the pia mater. In the remainder it was between the pia mater and the dura—i. e., in the arachnoid cavity. Hæmorrhages between the dura and the skull may be said never to occur except when associated with fracture. If the child is still-born, or, if death has occurred on the first or second day, the blood is partly fluid and partly coagulated; later it is entirely coagulated and may have undergone partial absorption. The amount of extravasated blood varies between one drachm and four ounces, the average amount being about one ounce. The blood extends into the fissures between the convolutions and sometimes into the ventricles along the choroid plexus, although this is rare. In large hæmorrhages the brain substance is softened and in places may be quite disintegrated; but with small extravasations these changes are very slight and hard to demonstrate to the naked eye, though causing remote consequences often of a serious nature. In cases which survive for two or three weeks there is usually a certain amount of meningitis. The later changes—those of arrested development of the cortex and cerebral sclerosis—will be considered in the chapter devoted to Cerebral Paralysis in the section on Diseases of the Nervous System. Hæmorrhages into the membranes of the upper part of the cord are found in a large proportion of the fatal cases. Associated hæmorrhages of the lungs and other organs are not uncommon.

**Symptoms.**—If the hæmorrhage is large, the child is usually still-born, although its movements may have been active up to the commencement of labour. When the hæmorrhage is not so large as to be immediately fatal, the child may show no symptoms except dulness or stupor, with feeble or irregular respiration, death following within the first twenty-four hours. A large proportion of the cases are born asphyxiated, and frequently they are resuscitated only after considerable effort. They nurse feebly or not at all. Convulsions are common in cases which last for four or five days, and more with hæmorrhages at the convexity than

with those at the base. Opisthotonus is often present, also general rigidity of the extremities, clenching of the hands, and increased knee-jerks. Rarely there is complete relaxation of all the muscles. Sometimes there are automatic movements. The respiration is usually disturbed; in most cases it is slow and irregular. The pulse is feeble and usually slow. The pupils are more frequently contracted than dilated, and there may be oscillation of the eyeballs. There may be a slight exophthalmos. In large hæmorrhages there is marked bulging of the fontanel, and often separation of the sutures. If the hæmorrhage covers one hemisphere, there is complete hemiplegia of the opposite side. Small localised cortical hæmorrhages may cause paralysis of the face, arm, or leg, according to the position of the lesion, or localised convulsions. In large hæmorrhages at the base convulsions are rare, and death occurs early, usually in the first two days. In extensive cortical hæmorrhages convulsions and rigidity of the extremities are frequent, and life may be prolonged indefinitely. There is usually no fever, but exceptionally the temperature may be high.

The majority of the fatal cases die within the first four days. In those lasting a longer time the symptoms are tonic spasm of the trunk, or of one or more of the extremities, with localised paralysis—monoplegia, diplegia, or hemiplegia, according to the lesion—and localised or general convulsions often continuing for two or three weeks and gradually subsiding. In the mildest cases nothing abnormal may be noticed until the child is old enough to walk or talk. In those more severe there may be gradual and continuous improvement of the early symptoms, and the case may go on to apparent recovery, but usually there is some permanent damage to the brain.

The main diagnostic symptoms in recent cases are: bulging fontanel, slow pulse, stupor, rigidity, increased reflexes, convulsions, and paralysis, especially when localised, and opisthotonus. These vary with the extent and situation of the lesion. Lumbar puncture has very doubtful value.

**Prognosis.**—A large hæmorrhage at the base quickly causes death; if it is located at the convexity, although the child may survive, there is always serious damage to the brain. Even from small hæmorrhages some permanent injury usually results, though the extent of this may not be evident for years.

**Treatment.**—This is mainly prophylactic, the chief indication being to shorten tedious labours by the early use of the forceps. Where the hæmorrhage has been attributed to the forceps, the damage has rather been the result of the long-continued pressure before they were used. Nothing can be done after delivery to limit the amount of the hæmorrhage, except to keep the child as quiet as possible. The removal of the clot by surgical operation has now been successfully accomplished by Cushing and others. With more accurate diagnosis there seems to be

no reason why a considerable number may not be saved. For the best results operation should be done as soon as possible. One great difficulty is that of early and accurate diagnosis. Paralysis whether localised or general is of greater value in diagnosis than are convulsions. The latter, however, are especially important when localised or continuous and threatening life. The operative risk, while considerable, is not to be measured against the permanent mental deficiency usually resulting in most of these children when nothing is done. Cases with similar symptoms are sometimes seen in which there is no extravasation of blood found at operation, but only intense congestion with an excessive serous exudate. In them also relief may follow operation. The hopeless outlook for such cases when not relieved, justifies the taking of great risks.

#### FACIAL PARALYSIS.

The usual cause of facial paralysis is the use of the forceps, but this does not explain all the cases. The etiology of those in which the forceps have not been used is still somewhat obscure. In peripheral facial palsy the nerve is pressed upon, either near its exit from the stylo-mastoid foramen, or where it crosses the ramus of the jaw, at which point the parotid gland gives it but little protection in the newly born. If the lesion is in front of this point, any one of the terminal branches may be affected; most frequently it is the temporo-facial branch. As only one blade of the forceps commonly touches the face in this region, the paralysis is, as a rule, unilateral.

Roulland has reported several cases not due to the forceps. In these the pressure is believed to have been produced by the promontory of the sacrum at the superior strait, or by the ischium at the inferior strait, as paralysis followed when the head was long arrested at one of these points. It was not seen with face or breech presentations. When facial paralysis is of central origin it depends generally upon a meningeal hæmorrhage, and the arm and leg of the same side as the face are involved. It is, however, possible for a very small cortical hæmorrhage to produce paralysis of the face only.

In repose, the only symptom noticed may be that the eye remains open upon the affected side, owing to paralysis of the orbicularis palpebrarum. When the muscles are called into action, as in crying, the whole side of the face is seen to be affected. The paralysed side is smooth, full, and often appears to be somewhat swollen. The mouth is drawn to the side not affected. In this paralysis, the tongue, of course, is not implicated. It is therefore rare that nursing is seriously interfered with.<sup>1</sup> If the paralysis is of central origin, only the lower half of the face is involved.

<sup>1</sup> In this connection it is to be remembered that the principal part in nursing is done by the tongue, and not by the lips.

while in peripheral paralysis, as the trunk of the nerve is injured, the upper half of the face, including the orbicularis palpebrarum, is also affected.

The paralysis is generally noticed on the first or second day of life, and does not increase in severity. Its course and termination depend upon the extent of the injury done to the nerve. Some idea of this may often be gained by the amount of injury to the soft parts, although this is not an infallible guide. In cases not due to the forceps, the paralysis is slight and disappears in a few days; the great majority of the forceps cases follow the same favourable course, the paralysis gradually disappearing without treatment in about two weeks. In more serious cases it may last for months, or it may be permanent. The reaction of degeneration is present in these severe cases, and there may even be perceptible atrophy of the muscles. This symptom is fortunately extremely rare.

**Treatment.**—Nothing should be done for the first ten days except to protect the eye and keep it clean. If improvement has begun by the end of this time, the probabilities are that the case will require no treatment. If no improvement has taken place by the end of the third or fourth week, electricity should be used regularly and systematically. If the muscles respond to it, the faradic current may be employed; if not, galvanism should be used. The electrical treatment should be continued for several months, or until recovery has taken place.

#### PARALYSIS OF THE UPPER EXTREMITY.

When this is due to a peripheral lesion it probably never involves the entire arm, but affects only certain muscles or groups of muscles. Although commonly occurring after an artificial delivery, it may be seen in cases where the labour has terminated naturally. Roulland has reported a case in which deltoid paralysis, occurring in a large child, was attributed to pressure upon the shoulder during labour. In vertex presentations, paralysis is most frequently due to the forceps where one of the blades has extended down upon the neck, injuring the lower cervical nerves. It may be produced by traction with the finger in the axilla. Roulland reports a unique case of paralysis of both extremities, apparently due to the cord being very tightly wound around the neck. The great proportion of all cases of paralysis of the upper extremity follow extraction in breech presentations. The injury is usually inflicted by traction upon the shoulder in the delivery of the head, or in bringing down the arms when they are above the head. In the latter case the paralysis may be double and associated with fracture of the clavicle or humerus. In shoulder presentations, paralysis may be produced by traction upon the arm itself. The primary lesion consists of an actual

rupture of nerve fibres and even of nerve trunks, probably with hæmorrhage into the nerve sheath. An inflammatory process follows as a result of which all these structures are fused together in one cicatricial mass.

The most common form of peripheral paralysis is that known as the "upper-arm type," or Erb's paralysis, in which the injury is inflicted at the anterior border of the trapezius muscle at the lower part of the neck, usually in such a position as to affect the fifth and sixth cervical nerves. The muscles paralysed are the deltoid, biceps, brachialis anticus, supinator longus, and sometimes the supra- and infra-spinatus. All these muscles may be involved, or only part of them, and in varying degrees. In case the injury is slight, the paralysis may not be noticed for some weeks. If severe, it is evident in the first few days. The arm hangs



FIG. 18.—ERB'S PARALYSIS.  
Infant two months old.

lifeless by the side; it is rotated inward, the forearm pronated, the palm looking outward (Fig. 18). The forearm and hand are not affected. In severe cases there may be anæsthesia of the outer surface of the arm, in the region supplied by the circumflex and external cutaneous nerves. This is rarely marked, and in its slighter degrees it is very difficult to determine. It is characteristic of this paralysis that the triceps is not affected, so that power to extend the forearm remains, although it cannot be flexed. Atrophy of the paralysed muscles occurs after a few weeks, but the muscles are so small and so covered with fat that it is rarely noticeable before the second year. It is most conspicuous in the deltoid. In all severe cases the reaction of degeneration is present. In some of the cases of long standing there occurs a shortening of the tendon of the subscapularis muscle, often associated with subluxation of the humerus. The paralysis may be complicated with fracture of the clavicle, the neck of the scapula, or the shaft of the humerus, or with epiphyseal separation of its head.

The *prognosis* depends upon the severity of the injury and also to some degree upon the time when treatment is begun. Many cases recover spontaneously in two or three months, improvement being observed within a few weeks, first in the biceps and last in the deltoid. Other cases

more severe in type recover after months as a result of systematic treatment by manipulation, massage, and electricity. The electrical reactions are of some value in prognosis. If the muscles respond to faradism, rapid improvement can generally be prophesied. If the reaction of degeneration is present, improvement will be slow and the paralysis is likely to be permanent. Permanent paralysis is most frequently of the deltoid.

The *diagnosis* is usually not difficult, since the great majority of cases are of the "upper-arm type" with classical symptoms. Peripheral palsy of the arm can scarcely be confounded with that of cerebral origin. If the lesion is central it is one of the rarest occurrences for the arm alone to be involved; either the leg or face, or both, are generally likewise affected. If the case does not come under observation until the child is a year old, it may be difficult, or without a good history it may be impossible to distinguish peripheral paralysis from that due to poliomyelitis. The peculiar group of muscles involved in Erb's paralysis is the only diagnostic point.

In recent cases the disability resulting from the tenderness or pain of syphilitic epiphysitis may simulate paralysis, but there is lacking the characteristic position of the arm, and a careful examination discloses the fact that the paralysis is only apparent. This may affect both sides. Fracture of the clavicle or epiphyseal separation of the head of the humerus may also be mistaken for paralysis. In cases of long standing, paralysis of the deltoid may resemble dislocation of the humerus. The reaction of degeneration differentiates paralysis from surgical injuries with similar deformities.

The *treatment* consists in the use of massage, manipulation, and electricity, which should be begun at the end of the first month, and used regularly and systematically for months. If the muscles respond to faradism this may be employed, but in most severe cases they do not, and galvanism must be used, according to the rules laid down for facial paralysis. For cases which do not recover either spontaneously or under treatment, or show no marked improvement before nine months, operation should be considered. This consists in dissecting out and suturing the nerve trunks whose continuity has been broken by the injury. A. S. Taylor, New York, from quite an extended experience, has reported marked improvement in some otherwise hopeless cases by this operation.

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## CHAPTER VII.

### TUMOURS OF THE UMBILICUS, MASTITIS, ETC.

**Granuloma.**—This is nothing more than a mass of exuberant granulations at the umbilical stump. The mass is generally about the size of a

pea—sometimes larger—bleeds readily, and has a thin, purulent discharge. It is promptly cured by the application of any simple astringent; powdered alum is probably the best. In case this is not successful, the granulations may be touched with nitrate of silver or snipped off with scissors.

**Adenoma, Mucous Polypus, or Diverticulum Tumour—Umbilical Fistula.**—The first three terms are used synonymously to describe an umbilical tumour covered with a mucous membrane which is similar in structure to that of the small intestine. It is usually associated with an umbilical fistula. This tumour is formed by a prolapse at the navel of the mucous membrane of Meckel's diverticulum. This diverticulum is the remains of the omphalo-mesenteric duct. When it is present in infants, it is found in various stages of development. Most frequently there is a blind pouch a few inches long given off from the lower part of the ileum. In other cases it may remain patent quite to the umbilicus, causing a fæcal fistula (Fig. 19, A). As the intestine below it is generally normal, this fistula may persist for months or even years, giving rise to no symptoms except a slight fæcal discharge from the umbilicus. In certain cases intestinal worms have been discharged through it. It may close spontaneously or be closed by operation.

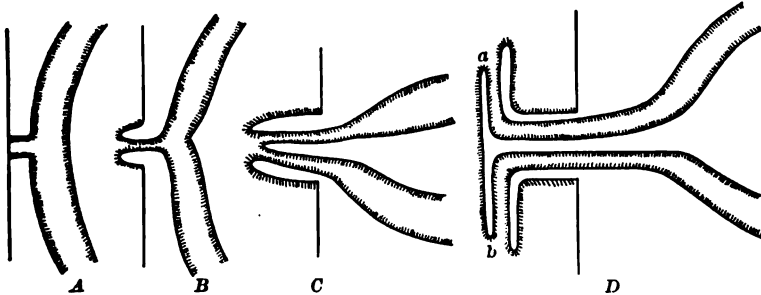


FIG. 19.—UMBILICAL FISTULA AND TUMOURS PRODUCED BY PROLAPSE OF MECKEL'S DIVERTICULUM. (Barth.)

A prolapse of the mucous membrane lining the diverticulum produces an umbilical tumour with a fistula at its summit (Fig. 19, B). This is the most common form. A cross-section shows under the microscope the structure of the intestinal mucous membrane both as an external covering and lining of the fistulous tract. The prolapse may involve not only the mucous membrane but the entire intestinal wall. There then exists a conical tumour with a fistula which has but one external opening, but at a short distance from the surface it bifurcates, one branch leading upward and one downward (Fig. 19, C). A continuation of the prolapse gives a broad pedunculated tumour (Fig. 19, D), which may reach the size of a man's fist. Its covering is the same as in the other forms.



It may contain several coils of intestine. In this form there are usually two fistulous openings (*a*, *b*) which communicate with the intestine.

In all of these cases the tumour is smooth, irreducible, of a rosy pink colour, and from its surface there oozes a mucous discharge. Microscopical examination shows the external covering to be the same in structure as the intestinal mucous membrane. These tumours are generally small, varying in size from a pea to a small cherry, but they may be very much larger. A fæcal fistula usually, but not invariably, coexists. In the condition represented in Fig. 19, B, it is easy to see how an obliteration of the fistula may occur. The small tumours are readily cured by the ligature. The larger ones are usually associated with other serious malformations of the intestines, which make the outlook bad in almost every instance.

#### UMBILICAL HERNIA.

Hernia into the umbilical cord is a rare congenital condition of a serious nature. It is due to some fœtal defect, and varies in size from a small protrusion to complete eventration in which nearly all the abdominal organs are outside the body. Many cases in which only intestinal coils are contained in the sac, though the tumour is quite large, are amenable to surgical treatment, which should be instituted at once. In the very large ones the prognosis is bad.

The common umbilical hernia is quite a different condition, and while a source of much annoyance it is rarely serious. It is much more common in females than in males, and occurs especially in those who are poorly nourished and rachitic. The tumour is usually from one-fourth to one-half an inch in diameter; it may, however, be very large, and may even become strangulated, when a surgical operation may become necessary. The ordinary cases, however, require only mechanical treatment. The most important thing is prevention. For this purpose it is necessary, after the cord has separated, to place a firm pad over the navel, and to use a snug abdominal band for the first two or three months. After this period it is uncommon for hernia to develop. In cases coming under observation after the third or fourth month, the pad and abdominal bandage are inadequate, and other means must be employed to retain the hernia. The best of these consists in the use of two adhesive strips applied obliquely over the abdomen, crossing at the umbilicus, the skin along the median line being folded inward so as to overlap the tumour, this forming the retention pad. A simple method of retention is to place over the tumour a coin or button covered with kid and hold it in position by a strip of adhesive plaster ten or twelve inches long. One should be cautious about using the small conical pads frequently employed, as these tend to dilate the opening rather than to close it. If the skin is made absolutely clean and zinc-oxide plaster used, excoriations are rare. The

dressing should be changed every few days and worn for several months. After the first year all mechanical treatment is unsatisfactory. For the very small tumours it is really unnecessary to use any form of apparatus, since these cases ordinarily show little or no tendency to increase in size, and the retention apparatus causes more annoyance than the hernia. These small herniæ sometimes disappear spontaneously during childhood, and rarely need be considered in children over seven years of age. Operation is seldom necessary.

## MASTITIS.

According to Guillot, a certain amount of secretion in the breasts of the newly born is physiological. It is certainly very common. It is most abundant between the eighth and fifteenth days, but may continue in small quantities as late as the third month. It is seen with equal frequency in both sexes. The quantity of the secretion amounts in most cases only to a few drops; in some, however, as much as a drachm has been obtained. Chemical analysis has shown this secretion to be essentially the same as the adult milk—containing fat, sugar, protein, and salts. In gross appearance it resembles colostrum. The researches of Sinéty have shown that the mammary gland of the newly born contains cul-de-sacs lined with secreting cells, resembling those of the adult. During the period of secretion the gland is slightly reddened, its vessels turgid, and all the signs of functional activity are present. This condition in itself is of no practical importance, and in most cases, if left alone, the secretion ceases spontaneously after a week or ten days. It sometimes happens, however, that the presence of this secretion tempts the nurse or attendant to rub or squeeze the breast. Such manipulation occasionally leads to serious results by exciting a mastitis which may terminate in abscess. Mastitis is not a very rare condition, and although the inflammation is not usually severe, it may be serious and even fatal. The predisposing cause is the congestion which accompanies functional activity, usually in the second week. The exciting cause is most often some form of traumatism—undue pressure, the squeezing of the breasts, or rough handling by the nurse. Through abrasions or fissures thus produced, micro-organisms find a ready entrance with the same result as in the adult. It seems possible that the germs may enter through the lactiferous ducts without any abrasion of the skin. Want of cleanliness is always a favourable condition for such infection.

The symptoms of mastitis usually begin during the second week of life. There are redness, swelling, and the usual signs of inflammation, which may terminate in resolution or in suppuration. The process may be limited to the mammary region, or a diffuse phlegmonous inflammation may be set up, and the case terminate fatally. In the female it is

possible for the cicatrisation which follows such an inflammation to interfere with the subsequent development of the gland. The general symptoms are restlessness, loss of sleep, disinclination to nurse, and loss of weight. In cases of diffuse phlegmonous inflammation the general symptoms are those of pyogenic infection.

Mastitis is usually due to want of cleanliness or traumatism; the parts should therefore be kept scrupulously clean, and on no account should squeezing of the breasts be permitted. They should be protected by a simple cotton pad. If acute inflammation develops, it should be treated in the beginning by hot applications. Should pus form, early incision with free drainage and general tonic and stimulant treatment are indicated.

#### INTESTINAL OBSTRUCTION.

The most frequent causes of intestinal obstruction in the newly born are malformations of the intestine; rarely it may be due to pressure from tumours, or from a persistent omphalo-mesenteric duct or artery. The various pathological conditions present in intestinal malformations are considered in the chapter on Diseases of the Intestines. The most common seat of obstruction is at the anus, the bowel being normally formed throughout, lacking only the external orifice. The next most frequent condition is obstruction in the rectum, which may be due either to a membranous septum in the gut, or to obliteration of the tube for some distance. These rectal obstructions are readily recognised. By the examining finger or a bougie the lower limit of the obstruction can be made out, but there is no means by which the upper limit can be determined except by opening the abdomen. When the obstruction is above the rectum, localisation is more difficult; but the most frequent seat is the duodenum. Of 38 cases collected by Gaertner, the seat of obstruction was the duodenum in 19 cases, the jejunum in 3, the ileum in 11, the colon in 6, the ileum and colon in 1. There is often obstruction at more than one point.

The symptoms vary with the seat and the degree of the obstruction. In atresia of the anus or rectum there is at first simply an absence of all discharges from the bowel. Later there is abdominal distention from dilatation of the sigmoid flexure and colon. After several days vomiting begins. If there is atresia of the duodenum or any part of the small intestine, vomiting begins early—usually by the second day of life—and it is persistent. Nothing is passed from the bowels after the first dark discharge of the contents of the colon, which is chiefly mucus. There is rapid asthenia, and death from inanition usually occurs in four or five days. The higher the obstruction the shorter the duration of life. If the condition is one of stenosis only, the symptoms are similar to those described but less severe, and life may be prolonged for several weeks, or even months. The constipation in these cases is not absolute. When the

cause of obstruction is external pressure, the symptoms do not always begin immediately after birth. I once saw a child in whom nothing abnormal was noticed for the first three weeks, but at the end of that time there developed all the signs of acute intestinal obstruction. Laparotomy revealed a loop of intestine constricted by a tiny cord, which was probably the remains of the omphalo-mesenteric duct.

Cases of imperforate anus and membranous septum in the rectum are readily relieved by proper surgical treatment. In the other varieties of obstruction, whether in the rectum, in the colon, or in the small intestine, although life may be prolonged by the formation of an artificial anus, the ultimate result is almost invariably fatal, death usually occurring from marasmus during the early weeks of life.

#### DIAPHRAGMATIC HERNIA.

This is due to a congenital deficiency in the diaphragm, which is usually on the left side. Of 118 cases collected by Livingston, 83 were on the left side, 18 on the right, 4 were central, 2 were double, in 1 the diaphragm was absent. With small openings only a single coil of in-



FIG. 20, A.—DIAPHRAGMATIC HERNIA OF THE RIGHT SIDE, POSTERIOR VIEW. Child sixteen months old.



FIG. 20, B.—THE SAME, IMMEDIATELY AFTER ADMINISTRATION OF BISMUTH IN SUSPENSION. Stomach in the right thoracic cavity.

testine, with large ones a considerable part of the abdominal contents, may be found in the thorax. This causes displacement of the heart, usually to the right side, prevents the full expansion of the left lung, and if the deformity occurs early in intra-uterine life the lung may remain rudimentary. If a large deficiency exists, infants may live but a few hours; with smaller ones, life may be prolonged indefinitely.

The symptoms noticed soon after birth are usually cyanosis, rapid respiration, a sunken abdomen, an overdistended chest and dyspnœa. Children often live but a few hours. In those who survive a longer time dyspnœa is generally the most prominent symptom. It may be constant, it may occur in severe paroxysms, or there may be attacks of cyanosis often of great severity, these being produced by an accumulation of gas in the stomach or the thoracic part of the intestine. Other symptoms may at times suggest intestinal obstruction. The physical signs vary much from time to time. Sometimes those of pneumothorax are present; at others there is so much dulness with the feeble respiratory sounds, as to suggest fluid. The signs are usually upon the left side, with displacement of the heart to the right. A positive diagnosis can be often made by means of the X-ray after the administration of bismuth. (See Figs. 20, A, and 20, B.) The condition is not amenable to treatment.

#### CONGENITAL STRIDOR.

This term has been given to a rather rare form of dyspnœa seen in very young infants, beginning usually in the first days of life. Respiration is noisy and inspiration is accompanied by a marked croaking, or crowing sound, and with recession of the soft parts of the chest wall, which, especially at times of excitement, may be very great, yet there is no cyanosis and no subjective distress. In spite of the apparent difficulty of respiration the child seems comfortable. Expiration is usually easy and voice and cry are normal. The stridor diminishes when the child is very quiet but usually does not quite disappear even in sleep.

The symptoms begin in most cases immediately after birth or during the first week or ten days of life. They may increase for three or four weeks, then remain about stationary until the sixth or eighth month; after which with the growth of the larynx the dyspnœa and stridor steadily diminish. By the end of the second year it is usually gone or heard only on occasion.

For our knowledge of this affection we are especially indebted to the observations of Thomson, of Edinburgh, who believes that the condition is primarily functional and due to a want of proper co-ordination of the respiratory muscles. Secondly there is produced a folding of the epiglottis upon itself along the median line, so that its lateral borders approximate each other. In many of the cases reported, however, the change in the larynx seems to be rather a malformation especially of the epiglottis, which greatly narrows the superior opening of the larynx. Congenital stridor is favoured by the soft collapsible character of the structures of the larynx in young infants and the strong suction force of inspiration.

The prognosis in most of these cases is good, the chief dangers being

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from intercurrent disease or from broncho-pneumonia. Considerable deformity of the thorax may be produced (pigeon breast) which may persist to later childhood.

The diagnostic features of congenital stridor are the noisy respiration with marked inspiratory dyspnoea and crowing, with the absence of distress or subjective symptoms of any kind. It seems to be more frequent in delicate children. Conditions with which it may be confounded are papilloma of the larynx, laryngismus stridulus, catarrhal croup, and laryngeal spasm associated with adenoids. The first three of these are excluded by the history and by the absence of changes in the voice; the last one by the fact that the child is not a mouth breather, that the dyspnoea is not increased by closing the mouth.

Congenital stridor is not amenable to special treatment. Should the dyspnoea reach an alarming degree tracheotomy may be performed. The indications are to maintain the child's general nutrition and to protect it, so far as possible, from diseases of the upper respiratory tract.

#### SCLEREMA.

Sclerema is a condition characterised by hardening of the skin and subcutaneous tissues. It may occur in circumscribed areas or extend over nearly the entire body. It affects infants who are very feeble and usually terminates fatally. Although sclerema is chiefly seen in the first days of life it is not limited to the newly born, but may occur at any time during the first few months. It is not to be confounded with oedema of the newly born, with which condition it is, however, sometimes associated. From published reports it appears to be of not very infrequent occurrence in Europe, chiefly in large foundling asylums. In America, sclerema is not a common disease. In the newly born, sclerema affects those who are premature or very feeble, sometimes those who are syphilitic. Later it may follow any condition leading to extreme exhaustion, especially the different forms of diarrhoeal disease.

The first thing to attract attention is usually the induration of the skin. It is often seen first in the calves or the dorsum of the feet, sometimes first in the cheeks, but soon extends over the greater part of the body. It is especially marked in the cheeks, buttocks, thighs and back, and regions where adipose tissue is abundant. It may affect the body uniformly or in circumscribed areas. The skin may be smooth or it may appear somewhat lobulated. The colour is normal or slightly bluish, often tinged with yellow. The lips are blue, and the capillary circulation so feeble that after pressure upon the nails the blood returns slowly or not at all. The limbs are stiff and board-like. The skin is cold to the touch, and often the thermometer in the axilla will not rise above 90° F. In one recorded case the axillary temperature was only 71° F.

The general feeling of the body has been well likened to that of a half-frozen cadaver. The tongue and the mucous membrane of the mouth are cold; no radial pulse can be felt; the respiration is slow, irregular, embarrassed, and at times the movements of the thorax are scarcely perceptible. The cry is a feeble whine, scarcely audible. The duration of the disease is usually from three to four days. Death occurs slowly and quietly. If recovery takes place there is gradual improvement in the circulation and nutrition, and, later, a disappearance of the areas of induration.

The causes of sclerema are general, the most important factors being loss of fluids, great feebleness with lowering of the body temperature, and, in consequence, hardening of the subcutaneous fat. There are no essential lesions in this disease. Atelectasis is often present, and may have something more than an accidental association, as incomplete aëration of the blood is no doubt a factor in the production of the symptoms. Microscopical examination has shown the skin to be normal in typical cases.

The prognosis is very bad, because of the grave conditions of which it is the expression, but it is not invariably fatal. In its milder forms, where treatment is begun early, recovery may take place. The diagnosis is to be made from œdema by the fact that there is no pitting upon pressure, by the rigidity of the body, and by the great reduction in the temperature. The most important thing in treatment is artificial heat; nothing but the incubator is efficient. In addition to this, care should be taken to promote the general nutrition by careful feeding and by all other means possible.

#### INANITION FEVER.

The term *inanition fever* is not altogether a satisfactory one; but, until these cases are better understood, it is adopted because it emphasises the very close connection which exists between the rise of temperature and the condition of inanition or starvation. Under this heading are included cases seen during the first five days of life—generally from the second to the fourth day—in which there is an elevation of temperature, apparently due to the fact that the infant gets very little, frequently nothing at all, from the breast at which it is being suckled. It is further characteristic of these cases that the temperature falls when the child is put upon a full breast, or when artificial feeding is begun, or even when water is administered, if freely given. Some have ascribed the symptoms to uric-acid infarction of the kidneys.

So far as my knowledge goes, the first to call attention to this condition was McLane (New York), who in 1890 reported to one of the medical societies an extraordinary case of hyperpyrexia in a newly-born

child. The infant was found on the sixth day with a temperature of 106° F., near which point it had remained for three days. The child was being suckled at a breast which was found to be absolutely dry. A wet-nurse was procured, the temperature fell to normal in a few hours, and the child, which when first seen was apparently in a hopeless condition, was soon perfectly well.

Since that time very extensive observations, extending to upward of three thousand cases, have been made at the Sloane Maternity and Nursery and Child's Hospitals, which have established the fact that a rise of temperature to 102° or even 104° F. is quite common in newly-born infants during the first few days. This fever is accompanied by no evidences of local disease, and ceases in nursing infants with the establishment of the free secretion of milk. The fall in temperature is often rapid, dropping to the normal in a few hours after having continued for three or four days, and in a large number of cases it does not rise again.

The following case is a fairly typical one of the more severe form: The patient was the second child, the first having died at the age of ten days, from no disease, it was said, but simply from exhaustion. At birth the infant, a boy, weighed eight and a quarter pounds and was apparently vigorous. During the first forty-eight hours his loss in weight was five and a half ounces and his condition good. I saw him on the evening of the third day. In the preceding twenty-four hours he had lost eight ounces in weight, and the temperature had gradually risen, until at the time of my visit it was 102.8° F. The body was limp, the child making no resistance to examination. He cried with a feeble whine; the restlessness of the early part of the day having given place to complete apathy. The lips and skin were very dry, the fontanel sunken, the pulse weak. As the father, a physician, expressed it, "he had been wilting through the day like a flower in the sun." Although put to the breast regularly, the child had apparently obtained very little. It was, in fact, impossible to squeeze any milk from the mother's breasts. Water was freely given and a wet-nurse secured in a few hours. The first milk was taken from the wet-nurse at 11 P.M., and the temperature, which fell gradually during the night, was normal the next morning and did not rise again. (See chart, Fig. 21.) During the succeeding four days the child gained eighteen ounces in weight, and at the end of a week was as well as an average infant of his age.

The symptoms are so uniform and so characteristic that they make for these cases of fever a class by themselves. The frequency with which this is seen is shown by the following statistics: Among 200 infants taken successively at the Nursery and Child's Hospital, 20 had fever during the first five days, reaching 101° F. or over, which was not explained by ordinary causes and followed the course above described. In



500 successive children born at the Sloane Maternity Hospital, there were 135 with a similar fever. It was seen in vigorous infants as well as in those who were delicate. The usual duration of the fever was

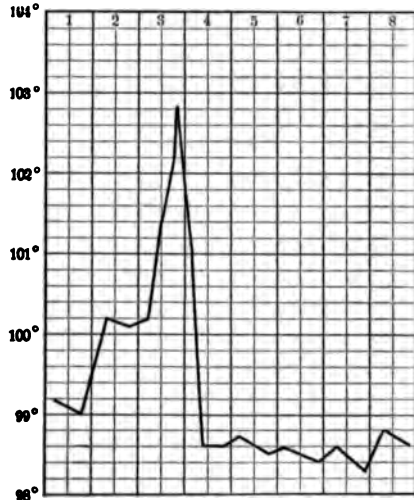


FIG. 21.—TEMPERATURE CHART. INANITION FEVER.

three days, the temperature generally touching the highest point upon the third or fourth day of life. In about two-thirds of the cases the temperature did not rise above 102° F.; in 9 it was 104° F. or over, the highest recorded being 106° F. The fall was generally quite abrupt, although not always so. Daily weighings, which were made in these cases, showed that the infants continued to lose weight while the fever continued, and that the loss almost invariably exceeded by several ounces that of the children who had no fever. The maximum loss noted was twenty-eight ounces. In quite a large number of cases it ex-

ceeded twenty ounces. As a rule the infants began to gain in weight when the temperature remained at the normal point, but not until then.

The symptoms presented by these infants were a hot, dry skin, marked restlessness, dry lips, and a disposition to suck vigorously anything within reach. With very high temperature there were considerable prostration and weakened pulse. In the less severe cases there were only crying and restlessness. The rapidity with which the symptoms disappeared when the children were wet-nursed or properly fed, was very striking.

It is important that this fever should be recognised, because it gives at times the first warning of a condition which may prove fatal. The extra loss of ten or fifteen ounces in the first week, is a serious handicap to newly-born infants, the effect of which may last for several weeks. The temperature of every child should be taken during the first week. All the usual local causes of fever are first to be excluded by a physical examination. This fever can hardly be confounded with that due to pyogenic infection, which rarely begins before the fifth or sixth day.

The treatment is simple, viz., to give water regularly every two hours, in quantities up to an ounce at a time if required by the thirst of the child. This should be done in every case where the temperature reaches 101° F. When the temperature does not at once begin to fall, the infant should be put upon another breast or artificial feeding should be begun.

Examination of the breasts from which the child has been nursing will usually reveal the fact that the secretion of milk is very scanty and often entirely absent.

Such a fever I have occasionally seen in older infants, usually in those who are nursing dry breasts or where fluid food and water have been withheld because of some gastric disturbance. It yields as promptly to treatment as does the same condition in the newly born.

SECTION II.  
NUTRITION.  
CHAPTER I.  
*INTRODUCTORY.*

NUTRITION in its broadest sense is the most important branch of pædiatrics. In no other field and at no other time of life does prophylaxis give such results as in the conditions of nutrition in infancy. The largest part of the immense mortality of the first year is traceable directly to disorders of nutrition. The importance of correct ideas regarding this subject can hardly be overestimated. The problem is not simply to save life during the perilous first year, but to adopt those means which shall tend to healthy growth and normal development. The child must be fed so as to avoid not only the immediate dangers of acute indigestion, diarrhœa, and marasmus, but the more remote ones of chronic indigestion, rickets, scurvy, and general malnutrition, since these conditions are the most important predisposing causes of acute disease in early life.

One of the difficulties has always been that temporary success may mean ultimate failure. If the injurious effects of improper feeding were immediately manifest, there would be very much less of it than exists at the present time. Many things are valuable as temporary foods, which when used permanently are injurious. No better illustration of this is seen than in the too exclusive use of the carbohydrate foods. Infants fed upon many of the proprietary foods often grow very fat, and for the time appear to be properly nourished. The effect of the absence from the diet of some of those elements which are of vital importance may not be evident for months. The physiological laws regarding the requirements of the growing organism cannot be ignored without serious consequences, which will sooner or later be evident. Correct ideas of infant feeding are based upon a knowledge of these laws. An accurate understanding of fundamental principles is essential to success and the vast majority of failures may be ascribed to ignorance or disregard of them.

THE FOOD CONSTITUENTS AND THE PURPOSES THEY SUBSERVE  
IN NUTRITION.

In infancy and childhood, as in adult life, the elements of the food are five in number: protein, fat, carbohydrates, mineral salts, and water. The forms in which they must be furnished to the child, and the relative quantities in which they are demanded, are different from those required by the adult. One reason for this difference is the delicate structure of the organs of digestion in infancy, and their inability to assimilate certain forms of food. Again, provision must be made not only for the natural waste of the body, but for its rapid growth, nearly trebling in size, as it does, during the first twelve months.

**Protein.**—Protein is essential to life, since it is the only kind of food which is capable of replacing the continuous nitrogenous waste of the cells of the body, upon which health depends. Protein is also indispensable for the growth of the cells of the body. In the adult only the requirements of repair are to be supplied. In the child a much larger amount is demanded to provide for growth. Protein should not be called upon to supply animal heat, although without the aid either of the fats or the carbohydrates, protein may sustain life for a considerable time; but in so doing a great excess of such food is required. Such a diet taxes severely the digestive organs and those of elimination. When, however, fats and carbohydrates are added to the food, only one-half or one-third as much protein is required to replace the nitrogenous waste, as in the case of an exclusive protein diet.

Of all the forms in which protein food may be furnished to the body, in proportion to its nitrogen content, milk taxes the digestive organs least. This fact is of the greatest importance and indicates the superiority of milk as a food, not only for the first year but throughout childhood. The most easily digested protein is that of woman's milk. Regarding the protein of cow's milk there is no doubt that the view formerly held that it was very difficult of digestion was erroneous. On the contrary under most conditions it is digested and absorbed with facility. During the first year milk furnishes all the protein that is needed for proper nutrition. During the second year meat, eggs, etc., may be advantageously added to the diet.

The digestion of the protein is begun in the stomach but is principally carried on in the intestines.

The protein molecule is a very complex one when compared with that of the fats or the carbohydrates. Growing out of this complexity of structure is the possibility of an immense number of side-products which may be formed by the splitting up of the protein molecule by digestive ferments, or by the numbers and varieties of bacteria found in the intestine. While the products of decomposition of the carbohydrates

are often irritating, those formed from the protein may at times be toxic and may be the cause of obscure and severe clinical conditions.

The prolonged use of a diet in which the protein is insufficient in amount or of an unsuitable form, produces a certain definite group of symptoms which are not always referred to their proper cause. In infants the most striking are anæmia, poor circulation, feeble muscular power, disinclination to exertion, and various functional nervous disturbances. Such children are often very fat. The vegetable protein cannot permanently take the place of the animal protein in the food of young infants.

**Fats.**—Fats are the most important source of animal heat, their caloric value being a little more than twice as great as that of the carbohydrates or the protein. They save nitrogenous waste. The fats and carbohydrates should be supplied in the food in such amount that the entire energy of the protein may be utilized for the growth and the nutrition of the cells of the body without being drawn upon to furnish animal heat. Fats increase the body weight. The large amount of fat stored up in the subcutaneous tissues in infancy is one of the best evidences of health.

The fats supply important elements needed for the normal development of the nervous system. This fact is probably connected with the large amount of fat of various forms which the nerve structures contain. It is a familiar clinical fact that functional nervous disorders are exceedingly common as a result of the long-continued use of foods low in fat. Many such disturbances commonly seen with rickets are regarded by some as a consequence of fat-starvation.

In the growth of bone the fats play an important rôle. The fatty acids formed in the intestine by the splitting up of the neutral fats of the food, combine with the insoluble salts of lime and magnesium and in this way, chiefly, these substances necessary for the growth of the skeleton are absorbed. Normal bony development, therefore, suffers if the food is low in fat.

The unabsorbed fats have a distinct value in preserving the proper consistency of the faecal mass. While neither the protein of milk nor the milk sugar appears as such in the stools of the nursing infant, fat is abundant. It forms normally from twenty to thirty per cent of the dry substance of the stool. The amount furnished to the infant is, therefore, considerably in excess of the needs of the body for nutrition. The use of this excess seems to be to increase the volume of the stool and to keep the mass so soft as to be easily expelled.

The amount of fat required in infancy is relatively much greater than in adult life. A well-nourished nursing infant weighing fifteen pounds actually receives about one-half as much fat as is allowed in a ration for an adult doing moderate work, who weighs ten times as much.

The form in which fat is supplied during the first year is the butter-fat of milk. There are marked differences in the fats of woman's and cow's milk, which to an important degree affect their digestibility. These are more fully considered later. Fats should be supplied liberally throughout childhood in the form of cream, eggs, butter, olive or cod-liver oil.


While it is evident from the foregoing that the fat requirements of the young child are great, it must also be remembered that in certain conditions even the normal amount of fat is badly borne and may do positive harm. Fats do not readily form products injurious to the economy as a consequence of imperfect digestion, but the amount given should be very greatly reduced in the following circumstances: (1) All wasting conditions depending upon disorders of digestion, whether due to functional derangement of the stomach, intestine, liver, or pancreas, or to chronic catarrhal inflammations of the stomach or intestine; (2) all acute disorders of digestion or acute inflammations of the stomach or intestines; (3) all febrile conditions, no matter from what cause; (4) during periods of very hot weather. A failure to regard these contraindications is a constant source of trouble in practice.

In the conditions just enumerated the fats must largely be replaced by the carbohydrates, as these substances are capable for the time being of assuming the functions of the fats, and besides are easily digested and assimilated. Such substitution should not be continued too long, as serious results may follow.

**Carbohydrates.**—Although these, like the fats, can not replace the nitrogenous waste of the body, they are important aids to the protein, and in this respect they are even more valuable than the fats. The carbohydrates are partly converted into fats, and may thus increase the body weight. They are capable of replacing the fat-waste of the body. They are one of the most important sources of animal heat.

Carbohydrates are the most abundant of the solid elements of the food, although they form a smaller percentage of the entire quantity of food in infancy than in adult life. The soluble carbohydrates which are used as foods for children are milk sugar, cane sugar, and maltose. Since all of these are converted by digestion into glucose they are to a certain degree interchangeable. In selecting milk sugar as the chief carbohydrate for the first year, we are following Nature, for this is what is furnished in the milk of all mammals. Milk sugar has a decided advantage in not fermenting with the common varieties of yeast present in the stomach, as do both maltose and cane sugar. Like the other sugars, however, milk sugar does readily undergo fermentation in the intestine by the action of bacteria.

The ability of the young infant to digest starches is relatively feeble, although this power does exist to some degree from birth; but the greater



part of the carbohydrates required should be furnished in the form of sugars. To infants of four months and over, starches may at times advantageously be added to the diet, and after eight months the quantity may be considerably increased. But in whatever form or quantity used thorough cooking is indispensable. Insufficient cooking is responsible for much of the starch indigestion seen in young children.

The advantages of the carbohydrates as foods depend upon their easy digestibility. The transformation of any of the sugars into glucose is a relatively slight chemical change, when compared with that which is necessary in the fats or protein before they can be absorbed.

The carbohydrates are at a great disadvantage on account of the readiness with which they undergo fermentation in different parts of the alimentary tract. To such fermentation are due many of the symptoms seen in the common functional disorders of digestion.

A diet consisting too exclusively of carbohydrates leads often to a rapid increase in weight, but it is not accompanied by a proportionate increase in strength. Infants so fed have but little resistance, and many of them become rachitic. The easy digestion of foods consisting chiefly of soluble carbohydrates, such as condensed milk and the proprietary infant foods, and the rapidity with which children so fed gain in weight, lead to a great misapprehension in regard to their value as foods. The ultimate results of such one-sided feeding, if long continued, are almost invariably disastrous.

In building up the cells of the body the protein is first in importance, the carbohydrates second, and the fats third. In the production of animal heat the fats come first, the carbohydrates second; practically the protein should never be called upon for this purpose. In a proper diet, all of these elements are represented.

**Mineral Salts.**—These are relatively of greater importance in infancy than in later life, because of the rapid development of the skeleton during infancy and early childhood. The most important for this purpose are the phosphates of lime and magnesium. These are furnished in abundance both in woman's and cow's milk; but are deficient in practically all the substitutes for milk. Salts are necessary for cell growth. They furnish the elements from which the mineral constituents of the blood and digestive fluids are formed, and facilitate absorption, excretion, and secretion. In fact, no function of the body is possible without the presence of salts in their proper proportions.

**Water.**—The food of all young mammals consists of from eighty to ninety per cent of water. This is needed for the solution of certain parts of the food, such as the sugar, the salts, and some of the protein, and for the suspension of other protein and the emulsified fat. All the food is thus dissolved or very finely divided so as to be more readily acted upon by the delicate digestive organs of the infant. Water is needed also in

large quantities for the rapid elimination of the waste of the body. In proportion to its weight, an average infant during the first year requires about five times as much water as an adult. During the time when the child is upon an entirely fluid diet, the addition of much water other than that supplied by the food is unnecessary; but when the number of feedings becomes less frequent, and solid food is given in larger quantities, water should be given freely between the feedings at all seasons, but especially in the summer.

**Caloric Values.**—The different foodstuffs have different caloric values:

|                                 |              |
|---------------------------------|--------------|
| One gramme of fat yields .....  | 9.3 calories |
| “ “ “ carbohydrate yields ..... | 4.1 “        |
| “ “ “ protein yields.....       | 4.1 “        |

It is important that these caloric values should be considered in the dietary of the child. The practical application of these facts is taken up in connection with the subject of infant feeding.

## CHAPTER II.

### THE INFANT'S DIETARY.

#### WOMAN'S MILK.

WOMAN'S milk is the ideal infant-food. A thorough knowledge of its character, exact composition, and variations is indispensable, for upon this knowledge are based all our rules for the preparation of foods used as substitutes for woman's milk when this can not be obtained.

Woman's milk is a secretion of the mammary glands and not a mere transudation from the blood-vessels; although under abnormal conditions it may partake more of the character of a transudation than a secretion. A few drops may be squeezed from the breasts before parturition; generally speaking, however, it is only present after delivery. During the first two days the secretion is scanty. Usually upon the third or fourth day it becomes well established, although it may be delayed several days longer and yet become abundant. During the period of lactation, milk is constantly formed in the mammary glands, but the process is more active while the child is at the breast.

**Physical Characters.**—Woman's milk is of a bluish-white colour and quite sweet to the taste. When freshly drawn its reaction is amphoteric to litmus, or slightly acid to phenolphthalein. The specific gravity varies between 1.026 and 1.036, the average being 1.031 at 60° F. On the addition of acetic acid only a slight coagulation is seen, this being in the form of small flocculi, and never in large masses as is the case



in cow's milk. Microscopically, there are seen great numbers of fat-globules nearly uniform in size and some granular matter. Occasionally there are present epithelial cells from the milk-ducts or from the nipple.

**Colostrum.**—The secretion of the first three or four days differs quite markedly from the later milk. To this the name *colostrum* has been given. It is of a deep yellow colour, which is chiefly due to the colos-

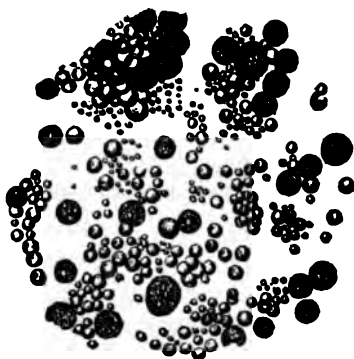


FIG. 22, A.—COLOSTRUM. (Funke.)



FIG. 22, B.—WOMAN'S MILK AT A LATE PERIOD. (Funke.)

trum-corpuscles. It is not so sweet as the later milk. It has a specific gravity of 1.030 to 1.040, a strongly alkaline reaction, and is coagulated into solid masses by heat, and sometimes coagulates spontaneously. It is very rich in protein and in salts. Microscopically the fat-globules are of unequal size, and there are present large numbers of granular bodies known as colostrum-corpuscles (Fig. 22, A). These are four or five times the size of the milk-globules (Fig. 22, B), and they are probably leucocytes in which are contained numerous fat granules. They are much larger than ordinary leucocytes and are nucleated.

*Composition of Colostrum.*

|               |        |
|---------------|--------|
| Protein ..... | 5.71   |
| Fat .....     | 2.04   |
| Sugar .....   | 3.74   |
| Salts .....   | 0.28   |
| Water .....   | 88.23  |
|               | <hr/>  |
|               | 100.00 |

The colostrum-corpuscles are very abundant during the first few days, but under normal conditions they are not found after the tenth or twelfth day.

**Daily Quantity.**—Exact information upon this point is difficult to obtain. There are recorded, however, extended observations made with

great care upon eight cases,<sup>1</sup> from which some deductions may safely be drawn. All were healthy infants, nursing exclusively and gaining steadily in weight.

From these observations, and others less extended, the average daily

<sup>1</sup> Hæhner's cases (Jahrb. f. Kinderh., xv, 23; xxi, 314). Case I. Female; birth-weight 7 pounds 14 ounces (3,100 grammes). First week, lost 1½ ounce (45 grammes); after this gained steadily during the twenty-three weeks of observation; from second to ninth week, average weekly gain 8 ounces (241 grammes); from tenth to eighteenth week, average gain 4½ ounces (138 grammes); from nineteenth to twenty-third week, average gain 4 ounces (130 grammes); weight at the end of twenty-third week, 14 pounds (6,690 grammes).

Case II. Male; birth-weight 6½ pounds (2,950 grammes). Loss, first week, 3 ounces (90 grammes); after this gained steadily during the eleven weeks of observation; from second to eleventh week, average weekly gain 7½ ounces (214 grammes); weight at end of eleventh week, 11 pounds 2 ounces (5,045 grammes).

Case III. Female; birth-weight 3 pounds 9 ounces (1,620 grammes). Gain, first week, 1½ ounce (45 grammes); during the succeeding twenty-one weeks of observation, average weekly gain 5 ounces (141 grammes); weight at the end of twenty-second week, 10 pounds 3 ounces (4,620 grammes).

Laure's case (Thèse, Paris, 1889). Female; birth-weight 8 pounds 13 ounces (4,000 grammes); loss, first week, 8 ounces (225 grammes); after this gained steadily during the nine weeks of observation, on an average 9½ ounces (268 grammes) weekly; at the end of ninth week, weight 13 pounds 3½ ounces (6,000 grammes).

Ahlfeld's case (Deutsch. Ztschr. f. Prakt. Med., 1878). Birth-weight 7 pounds 14 ounces (3,100 grammes). Observations continued from fourth to thirtieth week. During first ten weeks, average weekly gain 5½ ounces (161 grammes); from eleventh to twentieth week, 7½ ounces (214 grammes); from twenty-first to thirtieth week, 6 ounces (168 grammes); at the end of the thirtieth week, weight 18 pounds 9½ ounces (8,435 grammes).

Feer (Jahrb. f. Kinderh., xlii, 195). Three cases.

In all these cases the amount of milk was determined by weighing the infant both before and after every nursing during the entire period of observation. The following table gives in a condensed form the daily quantity of milk in these cases:

| TIME.                     | Hæhner's<br>1st case. | Hæhner's<br>2d case. | Hæhner's<br>3d case. | Laure's<br>case. | Ahlfeld's<br>case. | Feer's<br>3 cases.<br>Average. |
|---------------------------|-----------------------|----------------------|----------------------|------------------|--------------------|--------------------------------|
|                           | Grammes.              | Grammes.             | Grammes.             | Grammes.         | Grammes.           | Grammes.                       |
| 1st day                   | 20                    | 75                   | 20                   | ...              | ...                | ...                            |
| 2d day                    | 176                   | 135                  | 45                   | ...              | ...                | ...                            |
| 3d day                    | 265                   | 325                  | 70                   | 125              | ...                | ...                            |
| 4th day                   | 420                   | 295                  | 99                   | 222              | ...                | ...                            |
| 5th day                   | 360                   | 290                  | 124                  | 400              | ...                | 256                            |
| 6th day                   | 374                   | 340                  | 136                  | 475              | ...                | (average                       |
| 7th day                   | 423                   | 350                  | 156                  | 500              | ...                | 1st week)                      |
| Average 2d week           | 497                   | 423                  | 229                  | 556              | ...                | ...                            |
| Average 3d week           | 550                   | 468                  | 314                  | 730              | ...                | ...                            |
| Average 4th week          | 594                   | 531                  | 379                  | 810              | 576                | 610                            |
| Average 5th week          | 663                   | 561                  | 447                  | 944              | 655                | 667                            |
| Average 6th week          | 740                   | 661                  | 472                  | 978              | 791                | 753                            |
| Average 7th week          | 880                   | 681                  | 525                  | 1,038            | 811                | 802                            |
| Average 8th week          | 835                   | 730                  | 568                  | 1,024            | 845                | 815                            |
| Average 9th week          | 766                   | 665                  | 584                  | 1,085            | 810                | 820                            |
| Average 10th to 13th week | 796                   | ...                  | 600                  | ...              | 869                | 795                            |
| Average 14th to 17th week | 807                   | ...                  | 673                  | ...              | 983                | 845                            |
| Average 18th to 23d week  | 870                   | ...                  | 709                  | ...              | 1,029              | 919                            |
| Average 24th to 30th week | ...                   | ...                  | ...                  | ...              | 1,145              | 1,002                          |

quantity of milk secreted under normal conditions of health may be assumed to be pretty nearly as follows:

|   | Approximately.                    |
|---|-----------------------------------|
| At the end of the first week . . . . .          | 10 to 16 oz. (300 to 500 grm.).   |
| During the second week . . . . .                | 13 to 18 oz. (400 to 550 grm.).   |
| During the third week . . . . .                 | 14 to 24 oz. (430 to 720 grm.).   |
| During the fourth week . . . . .                | 16 to 26 oz. (500 to 800 grm.).   |
| From the fifth to the thirteenth week . . . . . | 20 to 34 oz. (600 to 1,030 grm.). |
| From the fourth to the sixth month . . . . .    | 24 to 38 oz. (720 to 1,150 grm.). |
| From the sixth to the ninth month . . . . .     | 30 to 40 oz. (900 to 1,220 grm.). |

It will be noted that the amount increases very rapidly up to about the eighth week, and after this much more slowly. The amount of milk varies also with the demands of the child in a very striking way.<sup>1</sup> The quantities mentioned can not be taken as an absolute guide as to the amount of food to be given to bottle-fed infants. Breast milk contains an average of twelve per cent solids; while the modification of cow's milk best suited to the early months seldom has more than from nine to eleven per cent solids. For this period, therefore, somewhat larger quantities are needed than of breast milk.

A comparison of the daily amount of milk taken with the weight of the child at the different periods, showed that both during the early and the later periods the larger children took not only more milk, but considerably more in proportion to their body weight than did the smaller ones. This harmonises with the common observation that small children are much more likely to be overfed than large ones.

The average quantity taken at one nursing by five of the children previously mentioned was as follows:

|  | Approximately.  |
|--|---|
| During the first week . . . . .                | $\frac{1}{2}$ to $1\frac{1}{2}$ oz. (18 to 45 grm.).    |
| During the second week . . . . .               | 1 to 3 oz. (30 to 90 grm.).                             |
| During the third week . . . . .                | $1\frac{1}{2}$ to 4 oz. (45 to 120 grm.).               |
| During the fourth week . . . . .               | $1\frac{1}{2}$ to $4\frac{1}{2}$ oz. (45 to 140 grm.).  |
| From the fifth to the seventh week . . . . .   | 2 to 5 oz. (64 to 150 grm.).                            |
| From the eighth to the eleventh week . . . . . | $2\frac{1}{2}$ to $5\frac{1}{2}$ oz. (75 to 160 grm.).  |
| During the fourth month . . . . .              | 3 to 6 oz. (90 to 180 grm.).                            |
| During the fifth month . . . . .               | $3\frac{1}{2}$ to $6\frac{1}{2}$ oz. (110 to 200 grm.). |
| During the sixth month . . . . .               | 4 to 7 oz. (120 to 220 grm.).                           |

Between the limits mentioned the greater number of cases will undoubtedly fall. The amount taken at one time is, however, modified by the frequency of nursing, and is therefore not so good a guide to the amount of food required, as is the quantity taken in twenty-four hours.

<sup>1</sup> There are a number of recorded instances in which the amount of milk secreted has been quite extraordinary—in some cases as much as four quarts daily. Lactation in exceptional instances also is unduly prolonged. I know of one well authenticated American case in which it continued for seven years. Among the Japanese it is frequent for it to continue up to three or four years. Among the Hottentots and other savage races lactation may be prolonged until the sixth or seventh year.

**Composition.**—Many of the older analyses of milk gave erroneous results because of imperfect methods of examination. According to the more recent analyses of Pfeiffer, Koenig, Leeds, Harrington, Adriance, and others, the composition of human milk is as follows:

|               | Normal average. | Common healthy variations. |        |
|---------------|-----------------|----------------------------|--------|
|               | Per cent.       | Per cent.                  |        |
| Fat .....     | 3.50            | 3.00 to                    | 5.00   |
| Sugar .....   | 7.00            | 6.00 "                     | 7.00   |
| Protein ..... | 1.25            | 1.00 "                     | 2.25   |
| Salts .....   | 0.20            | 0.18 "                     | 0.25   |
| Water .....   | 88.05           | 89.82 "                    | 85.50  |
|               | 100.00          | 100.00                     | 100.00 |

In the older analyses, the percentage of protein is almost invariably too high and the sugar too low.

The milk varies in composition somewhat with the period of lactation. That of the colostrum period is high in protein and salts and low in sugar. By the end of the second week all these elements have usually reached their normal averages. After this time until near the end of lactation the regular variations are slight. This is a point to be borne in mind in the selection of wet-nurses.

**Protein.**—The important forms of protein are casein and lactalbumin; several others, lactoglobulin, lactoprotein, and nuclein are also described. The casein is in suspension by virtue of the presence of lime phosphate in the milk, with which it is probably in combination. It coagulates only slightly with rennet, while acetic acid produces a loose flocculent precipitate. The lactalbumin resembles the serum-albumin of the blood. Chemists are by no means agreed in regard to the proportion of the different forms of protein present in milk. Lactalbumin exists in woman's milk in much larger amount than in cow's milk, and it is more abundant than the casein, the proportion of the two being, according to Koenig, about as five to four.

The total protein of normal milk is usually between one and two per cent. In abnormal specimens the variations are from 0.7 to 4.5 per cent. The protein is highest in the milk of the first few days; after the first month it varies but little until toward the close of lactation, when the amount falls very markedly.

**Fat.**—This exists in the form of minute globules, which are held in a state of permanent emulsion by the albuminous solution in which they are suspended. The fat of woman's milk is chiefly made up of the neutral fats—palmitine, stearine, and oleine; the last mentioned predominating. There are also small quantities of the fatty acids, but these are much less in amount than in cow's milk. In woman's milk the lower or volatile fatty acids are most abundant, while in cow's milk it is the higher fatty acids. Like the protein, the proportion of fat is subject

to wide variations, 3.5 per cent being taken as the normal average. In a series of thirty-four analyses made for me the fat varied between 1.12 and 6.66 per cent. The highest percentage I have known was 10.91. In forty-three analyses by Leeds, the variations were between 2.11 and 6.89 per cent. The proportion is very little affected by the period of lactation.

**Sugar.**—The sugar is in solution. Its proportion is nearly constant. The ordinary variations are usually within the limits of 6 and 7 per cent. The sugar being so important as a heat-producing element, Nature has wisely provided that this shall be the most constant ingredient of the milk. The amount of sugar is smallest in the milk of the first week; after the first month, however, the variations are slight.

**Salts.**—The average proportion of inorganic salts is 0.20 per cent, or a little more than one-fourth that of cow's milk. The proportion of the different salts is given in a subsequent chapter.

With the exception of calcium phosphate nearly all the salts are in solution. The milk of the first few days is very rich in salts; after the first month the variations are slight but show a gradual fall in the quantity present.

**The Examination of Milk.**—The exact composition of human milk is to be determined only by a complete chemical analysis. There are, however, many variations in composition which the physician may readily ascertain for himself by simple methods of examination.

The *quantity* of milk secreted by the breasts may be estimated by the quantity which may be drawn by a breast-pump, although this is not a very reliable test. If the child nurses habitually thirty or forty minutes, the probabilities are very strong that the quantity of milk is small. If the breasts at nursing time are full, hard, and tense, the supply is probably abundant. If the breasts are soft and flabby, and appear to fill only while the child is nursing, it is almost certain that the quantity is small. The most reliable of all tests is weighing

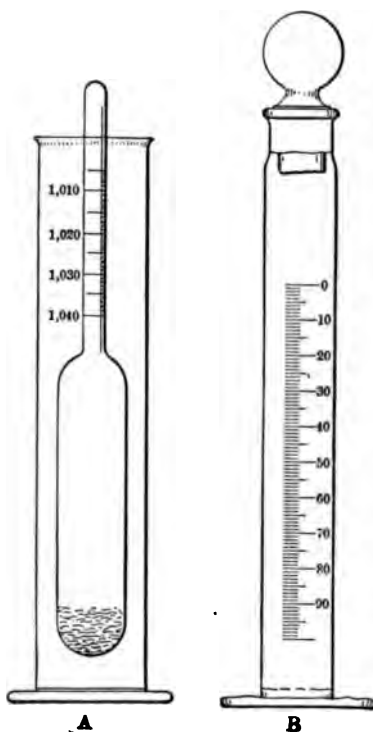


FIG. 23.—APPARATUS FOR EXAMINATION OF WOMAN'S MILK. The author's lactometer and cream-gauge.<sup>1</sup>

<sup>1</sup> The author's apparatus may be obtained from Eimer & Amend, Eighteenth Street and Third Avenue, New York. For a fuller discussion of the subject, see Archives of Pædiatrics, March, 1893.

the infant before and after nursing, upon an accurate pair of scales, sufficiently sensitive to indicate half-ounces. Two or three weighings will suffice to show conclusively whether an infant at three months, for instance, is getting habitually four or five, or only one or two ounces at a nursing.

The *reaction* of woman's milk even when freshly drawn is rarely alkaline, being amphoteric to litmus, or slightly acid to phenolphthalein.

The *specific gravity* may be taken with any small hydrometer graduated from 1.010 to 1.040 (Fig. 23, A). The specific gravity is lowered by the fat, but increased by the other solids. An ordinary urinometer will answer every purpose, the only difficulty being the quantity which is required to float the instrument.

*Microscopical Examination.*—The microscope may reveal the presence of fat globules, colostrum corpuscles, blood, pus, epithelium, and granular matter. Colostrum corpuscles are abnormal after the twelfth day; pus and blood are always abnormal. The presence of any of these elements necessitates the suspension of nursing, at least temporarily. But little importance can be attached to the size and appearance of the fat globules as affecting the nutritive properties of the milk.

*The Determination of Fat.*—The simplest method is by the cream-gauge (Fig. 23, B). Its results are only approximate, but in most cases sufficiently accurate for clinical purposes. The tube is filled to the zero mark with fresh milk, which stands, corked, at room temperature for twenty-four hours, when the percentage of cream is read off. The ratio of this to the fat is approximately five to three; thus 5 per cent cream indicates 3 per cent fat, etc.

For an accurate determination the best ready method is the Babcock test, which requires 20 c.c. of milk, or the modification by Lewi<sup>1</sup> of the Leffman and Beam test for cow's milk. This requires special tubes.

<sup>1</sup> Lewi's method is as follows:

(1) Place in the milk flask 2.92 c.c. of woman's milk measured in a special graduated pipette; (2) carefully rinse the pipette and add the same quantity of sulphuric acid C. P. of specific gravity 1.830. The acid should be added slowly, and mixed with the milk by gently rotating the flask. The colour turns to a very dark brown from the oxidation of the sugar and protein; (3) now add 0.6 c.c. of a mixture of equal parts of fusel oil and strong hydrochloric acid; (4) add sufficient of a mixture of the same sulphuric acid and water, equal parts, to bring the level of the fluid well up into the neck of the flask; (5) centrifuge for three or four minutes. The percentage of fat is now read off, each one-tenth gradation in the neck of the flask representing 0.3 per cent of fat in the specimen of milk.

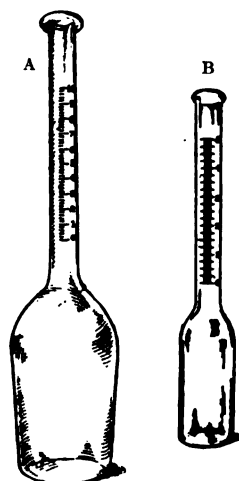


FIG. 24.—TUBES FOR DETERMINING THE FAT IN MILK. A, Babcock's tube for cow's milk; B, Lewi's modification for woman's milk.

*Sugar.*—The proportion of sugar is so nearly constant that it may be ignored in clinical examinations.

*Protein.*—Clinical methods for the estimation of the protein are not altogether satisfactory. The one giving the best results is that in which the protein is precipitated by a solution of phosphotungstic and hydrochloric acids in the Esbach tube, the percentages being read off after standing twenty-four hours.<sup>1</sup> We may also form an approximate idea of the protein from a knowledge of the specific gravity and the percentage of fat, if we regard the sugar and salts as constant, or so nearly so as not to affect the specific gravity. We may thus determine whether it is greatly in excess or very low, which, after all, is the important thing. The specific gravity will then vary directly with the proportion of protein, and inversely with the proportion of fat, i. e., high protein, high specific gravity; high fat, low specific gravity. The application of this principle will be seen by reference to the accompanying table.

*Woman's Milk.*

|                      | Specific gravity 70° F. | Cream—24 hours.  | Protein (estimated).       |
|----------------------|-------------------------|------------------|----------------------------|
| Average .....        | 1.031                   | 7%               | 1.5%                       |
| Normal variations... | 1.028–1.029             | 8%–12%           | Normal (rich milk)         |
| Normal variations... | 1.032                   | 5%–6%            | Normal (fair milk)         |
| Abnormal variations. | Low (below 1.028)       | High (above 10%) | Normal or slightly below   |
| Abnormal variations. | Low (below 1.028)       | Low (below 5%)   | Very low (very poor milk)  |
| Abnormal variations. | High (above 1.032)      | High             | Very high (very rich milk) |
| Abnormal variations. | High (above 1.032)      | Low              | Normal (or nearly so)      |

Any specimen taken for examination should be either the middle portion of the milk—i. e., after nursing two or three minutes—or, better, the entire quantity from one breast, since the composition of the milk will differ very much according to the time when it is drawn. The first milk is slightly richer in protein and much poorer in fat. The last drawn from the breasts is low in protein and high in fat. The following analyses from Forster illustrate these differences:

|               | First portion. | Second portion. | Third portion. |
|---------------|----------------|-----------------|----------------|
|               | Per cent.      | Per cent.       | Per cent.      |
| Fat .....     | 1.71           | 2.77            | 5.51           |
| Protein ..... | 1.13           | 0.94            | 0.71           |

**Conditions Affecting the Composition of Woman's Milk.**—*The Age of the Nurse.*—This has no constant influence. Other things being equal,

<sup>1</sup>For description see Boggs, Johns Hopkins Hospital Bulletin, No. 187, October, 1906.

the milk of very young women, and also of those over thirty-five years of age, is likely to be lower in fat than that of women between twenty and thirty-five years.

*Number of Pregnancies.*—Adriance found that the average milk of 23 primiparæ and 23 multiparæ, both taken at the third month, showed the following differences: The milk of the primiparæ was higher in fat (4.06 against 3.67) and in protein (1.61 against 1.35), but a little lower in sugar (6.52 against 6.85).

*Acute Illness.*—In the majority of cases of acute illness of a minor character and of short duration there is no perceptible effect upon the milk. In the acute febrile diseases of a severe type the quantity of milk is reduced, the fat is low, and the protein is apt to be high. In septic conditions bacteria may appear in the milk.

*Menstruation.*—The effect of this is exceedingly variable, depending much upon the individual and the ease of menstruation.

The nature of the changes in milk sometimes produced by menstruation is illustrated by the following case taken from Rotch:

|               | Second day of menstruation. Child's stools loose. | Seven days after menstruation. Bowels regular. | Forty days later. Child gaining rapidly. |
|---------------|---|--|--|
|               | Per cent.   | Per cent.                                      | Per cent.                                |
| Fat .....     | 1.37  | 2.02   | 2.74                                     |
| Sugar .....   | 6.10  | 6.55   | 6.35                                     |
| Protein ..... | 2.78  | 2.12   | 0.98                                     |
| Salts .....   | 0.15  | 0.15   | 0.14                                     |
| Water .....   | 89.60   | 89.16  | 89.79                                    |

From observations upon 685 cases, Meyer noted disturbances in the child in over one-half the number. My own experience accords rather with that of Pfeiffer and Schlichter, who consider it quite exceptional for the child to be visibly affected. Schlichter made observations upon infants during 233 menstrual days, noting the condition of the stools and digestion both before and after menstruation. In ninety per cent of the cases there was no perceptible influence. In only eight per cent were the stools bad, and in only three per cent was there disturbance of the stomach with vomiting.

At the present time sufficient observations have not been made to show whether the differences noted in the case cited above—low fat and high protein—are the rule where disturbances are produced during menstruation. Monti's examinations lead him to the conclusion that the fat is not constantly affected. It is safe to say that the changes are not uniform, and that in very many cases none of importance are produced by menstruation.

*Diet.*—The fat and the protein of the milk are much influenced by diet, the sugar but very little. The fat is increased by overfeeding es-



pecially with fats and carbohydrates, with little or no exercise; it is reduced by stopping these articles and substituting vegetables and by increasing the amount of exercise. The protein is increased by overfeeding and also by too little exercise. Starvation lowers the fat and sometimes also the protein. All fluids tend to increase the quantity of milk. Alcohol in the form of malted drinks, and malt extracts increase the quantity of milk and the amount of fat. The effect of alcohol upon the protein is not constant, but it is usually increased. The following table gives the result of analyses of the milk of two women observed in the New York Infant Asylum before, while taking, and after taking an alcoholic extract of malt:

|                 | I.<br>Without malt. | II.<br>After taking 8 os. malt<br>daily for 10 days. | III.<br>No malt for 7 days. |
|-----------------|---------------------|--|-----------------------------|
| <b>Case I:</b>  | <b>Per cent.</b>    | <b>Per cent.</b>                                     | <b>Per cent.</b>            |
| Fat .....       | 1.74                | 3.83   | 2.41                        |
| Protein .....   | 1.93                | 1.58   | 2.95                        |
| Sugar .....     | 7.02                | 7.43   | 6.59                        |
| Salts .....     | 0.20                | 0.17   | 0.19                        |
| <b>Case II:</b> |                     |  |                             |
| Fat .....       | 1.12                | 2.75   | 1.70                        |
| Protein .....   | 1.57                | 2.34   | 1.26                        |
| Sugar .....     | 7.11                | 6.77   | 6.04                        |
| Salts .....     | 0.19                | 0.17   | 0.18                        |

The child of Case I gained one ounce and a half during the four days preceding the first analysis; that of Case II did not gain at all. During the ten days while taking the malt, the first child gained twelve ounces, the second child eight ounces. During the seven days after the malt was discontinued, the first child gained eight ounces, the second child one ounce. There was a notable increase in the quantity of milk in both cases while taking the malt.

The nursing woman should have a generous diet of simple food, and should drink largely of milk or gruels made with milk. The diet should be a varied one, not excessive in nitrogenous food nor in vegetables. All salads, pastry, and highly seasoned dishes should be avoided, not so much because they upset the child, although this may happen, as because they are likely to disturb the digestion of the nurse. Nearly all the common vegetables and sweet fruits in season may be allowed in moderation. Strong tea and coffee should be prohibited, although weak tea or coffee may be allowed, each but once a day. Cocoa is not objectionable. In addition to her regular meals the nurse should have milk or gruel at bedtime. The diet should in all cases be adapted to her digestion. The bowels should move daily, by the use of laxatives if necessary. Great harm often results from overfeeding with its consequent indigestion. Alcoholic beverages should be forbidden.

**Drugs.**—The elimination of drugs through the milk is somewhat uncertain and variable; few of those popularly supposed to affect the child through the milk really do so. Given in full doses, belladonna regularly appears in the milk. Opium does not do so constantly; but when the milk is poor, enough may be excreted to produce serious symptoms. The iodides and bromides when long administered may be eliminated in sufficient quantity to produce their constitutional effects in the child. Mercury does not appear regularly, but only after prolonged use, and then in variable quantity. Most of the saline cathartics, arsenic, and the salicylates are occasionally found in the milk. Alcohol may seriously disturb the child if taken in considerable quantities by a nurse, although its elimination through the milk is doubtful.

**Pregnancy.**—The milk of pregnant women is generally scanty and poor in quality, especially in fat. (See Weaning.)

**Bacteria.**—Under normal conditions woman's milk may contain a few bacteria. They are chiefly cocci derived from the external milk ducts and are of no importance. In suppurative inflammation of the mammary gland, numerous bacteria may be found in the milk; also in some cases of puerperal sepsis. Tubercle bacilli have been demonstrated by Roger and Garnier in the milk of a woman with advanced tuberculosis, but ordinarily they are not present unless the gland is the seat of the disease.

**The Elimination of Antitoxin and Other Protective Substances by the Milk.**—The immunity of nursing infants to most of the contagious diseases has long been noted, but until recently little understood. Animal experiments have demonstrated the constant presence of diphtheria antitoxin in the milk of immunised animals. The Widal reaction has been obtained with the milk of mothers suffering from typhoid and with the blood of their healthy nursing infants.

**Nervous Impressions.**—The effect of the nervous condition of a woman upon her milk secretion is very striking, and much more important than the diet. Both the quantity and the composition of the milk are markedly changed by many different nervous impressions. Fright, grief, passion, excessive sexual indulgence, or any great excitement may entirely arrest the secretion, or if not arrested the milk may be so altered in composition as to make the child acutely ill. Worry, anxiety, fatigue, intense or prolonged nervous strain may so alter the milk as to cause it to disagree with a child who had previously thrived well upon it, or they may greatly diminish and sometimes even arrest the secretion. It is the nervous condition of the mother more than anything else which determines her success or failure as a nurse. If a mother would nurse successfully, she must have plenty of rest and sleep, moderate exercise, keep her mind free from unnecessary worries, avoid social engagements, and lead a simple, regular, natural

life. Unless she can and will do this successful nursing can hardly be expected.

The nature of the changes produced in milk by nervous disturbances in the mother are as yet little understood. Some infants are so profoundly affected as to suggest the development of toxic substances in the milk. The milk of the tired and worried mother is nearly always low in fat, while the protein is usually high, and possibly there are other changes as yet unknown.

#### COW'S MILK.

The only one of the lower animals whose milk is practically available for infant feeding is the cow. Cow's milk being our main reliance in the artificial feeding of infants and the staple food of nearly all young children, it follows that everything relating to its production and handling is important. The practising physician should therefore familiarise himself with the main facts regarding the production and handling of milk according to modern methods, since no one can do more than he to educate public opinion in these matters, and so to improve the milk supply of the community. Only an outline of the subject can be presented here. For more minute knowledge the reader is referred to special works upon the subject.<sup>1</sup>

The essential conditions to be fulfilled in cow's milk which is to be used as a food for infants and young children are: (1) Freshness; (2) it should contain no preservatives; (3) it should be from healthy animals, free from tuberculosis or other taint; (4) it should be clean; (5) it should not be skimmed or otherwise falsified; (6) it should contain no pathogenic organisms; (7) the number of other organisms should not be excessive. It is also desirable for purposes of infant feeding that the composition of the milk, particularly the percentage of fat, should be known, and that the milk should be as nearly uniform as possible from day to day and at different seasons of the year. Mixed or herd milk is therefore to be preferred to that from a single animal, since it is subject to fewer variations. The common varieties or "grade cows" should be chosen rather than highly bred animals, if for no other reason, because they are more hardy, less subject to disease, and less susceptible to other influences which might affect the milk.

As ordinarily handled, milk should be used before it is twenty-four hours old; after this time changes occur very rapidly, and such milk can not in summer be used with safety for young children. Milk may

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<sup>1</sup> Convenient works for a physician's use are Richmond's Dairy Chemistry; Aikman's Milk, Its Nature and Composition, Black, London; Russell's Outlines of Dairy Bacteriology; Belcher's Clean Milk, Hardy Publishing Co., New York; Pearsons' Jensen's Milk Hygiene, Lippincott Co.; Milk and Its Relation to Public Health, Bulletin 56, U. S. Public Health and Marine-Hospital Service.

be safe when more than twenty-four hours old provided special precautions are taken regarding cleanliness in producing and handling it, and special care in keeping it constantly at a temperature below 50° F.

Preservatives are very often added, particularly in hot weather, by unscrupulous dealers to retard the souring of milk, in order thereby to avoid the necessity and expense of proper icing. Formerly boric or salicylic acid was, and recently formaldehyd has been largely employed for this purpose.

**Micro-organisms in Milk.**—Most of the common bacteria grow readily in milk, and the conditions under which it is produced and handled render it liable to contamination in many ways.

1. *Disease in the Cow.*—From disease of the udder streptococci or other pyogenic germs may enter the milk in such numbers as to excite acute gastro-enteritis in a child. Other diseases which may be communicated from the cow are tuberculosis, anthrax, and the foot-and-mouth disease. In the State of New York it is estimated that 7 per cent of the cows are tuberculous. Pearson and Ravenel estimate the proportion in Pennsylvania at 2 or 3 per cent, while Marshall states that from 10 to 25 per cent of the Eastern dairy cattle are tuberculous. The best veterinarians regard tuberculosis as steadily increasing among cattle in the United States, particularly in the Eastern States. Of the cattle slaughtered in London, 25 per cent are stated to be tuberculous. Unless the process is advanced or the udder is the seat of disease, very many tuberculous cows do not have tubercle bacilli in their milk. Nevertheless tubercle bacilli are frequently found in ordinary market milk. In 107 unselected specimens of milk sold from cans in New York City Hess found tubercle bacilli in 17, or 16 per cent. Rabinowitch and Kempner in 25 similar examinations in Berlin found tubercle bacilli in 7, or 28 per cent. Macfadyen in London found, in 77 samples of milk, tubercle bacilli present in 17, or 22 per cent. These figures may be taken to represent average conditions in large cities. But the dangers from milk are not quite so great as would appear from these findings, for in many of the cases the number of bacilli is very small and only discovered by animal inoculations.

For reasons given elsewhere (*vide* Tuberculosis), I can not believe the danger of acquiring tuberculosis through milk as great as many have represented. For the present milk must be regarded as one of the possible sources of tuberculous infection. The sale of milk from cows showing evidence of tuberculosis upon physical examination, and from those having tuberculosis of the udder should not be permitted. Whether we should go further and exclude also the milk of every cow which reacts to the tuberculin test is still an open question.

2. *Specific Pathogenic Organisms Accidentally Gaining Access to Milk.*—The rôle of milk in the spread of infectious disease may be ap-

preciated by the fact that in 1900 Kober collected records of 330 outbreaks which were traced to it. The most important disease communicated in this way is typhoid fever. In the reports of 195 epidemics collected, typhoid existed at the dairy in 148 instances; in 67 the milk was diluted with infected well-water; in 7 the cows probably waded in polluted water; in 24 cases the employees acted as nurses to typhoid patients, and in 10 they continued at work, although themselves suffering from the disease; in one case it was found that the milk-pans were washed with cloths used about patients.

Next to typhoid the disease most often spread through milk is scarlet fever. A very small percentage of the cases of scarlet fever, however, can be traced to contaminated milk; but the sudden and simultaneous development of a considerable number of cases of this disease in a community should lead one to consider the milk supply as a possible cause. Of 99 epidemics of scarlet fever, there was disease at the farm or dairy in 68; in 17, employees were themselves affected, and in 10 they acted as nurses; in 6, persons connected with the dairy either lodged in or had visited infected houses; in 2 infection was brought by cans or bottles from the houses of patients; in 3 the milk was stored near or in the sick-room; in one case milk-utensils were wiped with an infected cloth.

Very infrequently diphtheria has been spread through milk. Of 36 outbreaks of diphtheria collected, there was disease at the farm or dairy in 13; in 3, employees themselves were ill. Twelve of the outbreaks included in this series, however, were of very doubtful character. Besides these diseases mentioned, cholera, dysentery, and certain forms of diarrhoeal diseases may probably be spread by milk.

3. *Other Bacteria Found in Milk.*—These are chiefly derived from the dust of the stable, the hands and clothing of the milker, and from the dirt which falls from the udder, belly, and tail of the cow into the pail during milking; very many come from the cow's excreta. Freeman exposed a Petri gelatin-plate beneath a cow's udder for one minute during milking and obtained 4,450 colonies. The varieties of bacteria found in fresh milk are many and vary with locality. Toward the souring point the great majority are of two or three varieties only; fully 95 per cent at that time belong to the lactic-acid-producing group. They cause the ordinary souring of milk by acting upon the milk sugar. Colon bacilli are very common. Other bacteria act upon the milk protein, inducing various putrefactive changes; and still others have a peptonising power.

Many of the bacteria are no doubt harmless. Others, while not, strictly speaking, pathogenic, when present in large numbers induce changes in milk that so impair its nutritive properties as to render it unfit for food, and in susceptible infants may cause serious illness. The

effects of bacterial contamination of milk are considered in the introductory chapter upon Diarrhoeal Diseases.

*The Number of Bacteria in Milk.*—This depends upon three conditions: (1) Cleanliness in handling; (2) temperature; (3) age of the milk. Hence the bacterial count becomes of the greatest value in furnishing information as to these matters, although of less importance in regard to the production of disease than the nature of the organisms present. The influence of the different factors may be illustrated by the following experiments made at the laboratory of the New York Health Department: A sample of milk taken under good conditions contained immediately after milking 300 bacteria in each drop. It was cooled to 45° F., and kept at this temperature. After twenty-four hours it contained in each drop only 200 bacteria; after forty-eight hours, 900; and after seventy-two hours, 150,000. The milk curdled on the sixth day. Another sample, taken in a dirty barn, cooled and kept at 52° F., contained at first 2,000 bacteria in each drop; in twenty-four hours, 6,000; in forty-eight hours, 245,000; in seventy-two hours, 16,500,000. The milk curdled on the fourth day. The influence of temperature alone upon the multiplication of bacteria in milk is well shown by the following experiment: Four samples of the same milk were kept at different temperatures for twenty-four hours and equal quantities were then plated; No. I was kept at 60° F. and showed 134,340 colonies; No. II was kept at 55° F. and showed 67,170; No. III was kept at 50° F. and showed 1,362; No. IV was kept at 45° F. and showed 448.

The number of bacteria in bottled milk from single dairies usually ranges from 10,000 to 100,000 per cubic centimetre, according to the season. Milk from mixed dairies delivered in cases ranges from 100,000 to 5,000,000, the latter number being often reached in very hot weather. There seems, however, to be little doubt that milk, in common with other animal fluids, possesses certain bactericidal properties which render it stable for a limited time, which are soon exhausted if the temperature is allowed to rise, but which assist materially in its preservation during the first twenty-four hours.

The number of bacteria in cream is nearly always greater than in milk. Freeman's experiments with gravity cream showed that the bacteria were 300 times as numerous in the cream as in the milk left behind, the bacteria being apparently carried up with the fat globules. This emphasises the necessity of the greatest care with reference to cream and indicates one great advantage of centrifugal cream, that it can be marketed at least twenty-four hours earlier than gravity cream.

*A Bacteriological Standard for Pure Milk.*—There has been much discussion among different milk commissions regarding some such standard. One commission requires that the milk shall not have more than 10,000 bacteria in each cubic centimetre; another fixes the limit

at 30,000. Methods of cultivating and counting the bacteria of milk are by no means uniform, and it is often quite impossible to compare the figures of different observers, because not all the conditions were the same. Too much stress may be laid upon the mere number of bacteria; their character must also be considered. A milk commission should be satisfied if all pathogenic organisms have been excluded and if the number of other organisms is below 30,000 per cubic centimetre. There is no evidence that when these conditions have been fulfilled the results in infant feeding are any better with a milk containing 5,000 bacteria or less, than with one containing 30,000. Nor is there any proof that milk containing 30,000 bacteria per cubic centimetre is for this reason injurious. Emphasis should be placed rather upon the hygienic conditions under which the milk is produced and the exclusion of pathogenic organisms. A low bacterial count, if no preservatives have been used, may be taken as presumptive evidence at least that the milk is produced under hygienic conditions and carefully handled, and under such circumstances the entrance of pathogenic germs is extremely improbable. It is quite possible to produce milk which is practically sterile; but the expense entailed is so great as to make the commercial production of such milk impracticable. For milk sold in cans 100,000 to the cubic centimetre may be considered good; for bottled milk anything under 30,000 is good, and an average under 10,000 is exceedingly good; the count in all cases being made at the time the milk is offered for sale.

The reports made by the bacteriologist of one of the New York milk commissions show that by the most careful handling the number of bacteria<sup>1</sup> can be kept at an average of less than 5,000 in each cubic centimetre at the time when it is delivered.

*The Means of Excluding Pathogenic Bacteria, and of Checking the Spread of Contagious Diseases through Milk.*—Rules are readily deducible from a study of the records of how milk has usually been infected.

1. No person suffering from, or in contact with a person suffering from, a contagious disease should enter a dairy building or in any way come in contact with the milk or milk-utensils; especially should this rule be enforced in the case of diphtheria, scarlet and typhoid fevers.

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<sup>1</sup> To accomplish such a result certain special precautions are observed; the most important are the following: The stables have cement floors to admit of ready flushing with a hose; no hay, straw, or fodder is kept in the stables; shavings are used for bedding; the cows are carefully groomed every day and not fed until after they are milked; a few minutes before milking the loose dirt is removed from the udders with a damp cloth. The milkers wear sterilised coats and caps, and wash their hands before milking each cow; all bottles, pails, etc., are sterilised with live steam, the pails just before using. The milk is immediately removed to the milk-house, where it is strained, mixed, cooled to 38° F., bottled and sealed—all within twenty minutes from the time it leaves the cows.

Children, domestic animals, and, so far as possible, flies should be excluded from rooms where milk is handled.

2. Milk should not be handled in or near dwellings, privies, or stables; cans and pails should be washed only at the dairy, and after ordinary cleansing they should be washed in boiling water or sterilised with live steam. Especial attention should be given to milk-bottles which have been in infected rooms. The hands of the milker should invariably be carefully washed just before milking.

3. Dairies should be subject to regular city or state inspection. Milk from cows showing physical evidence of tuberculosis should be excluded; also that from animals which are in any way sick or are suffering from disease of the udder. Milk from apparently healthy animals who respond to the tuberculin test should not be used for food in a raw state.

4. In any epidemic of contagious disease, the milk supply should be carefully investigated; and all cases of such diseases in the families of those who produce or handle the milk should be immediately reported and supervised by the authorities.

*Means of Reducing the Number and Lessening the Growth of Bacteria in Milk.*—A marked diminution in the number of germs present in milk, as it is now handled, may be brought about by attention to two conditions—cleanliness and temperature—and the results will be directly in proportion to the care bestowed upon them.

Cleanliness must have reference, in the first place, to the cows themselves. Since most of the germs in milk come from the cows, it is important that the belly, udder, and tail should be cleansed before milking, to prevent droppings into the pail. The parts should be wiped with a damp cloth. Milking should be done out of doors or in a clean, special shed; if in the stable, this should be clean. No dry fodder should be fed and no sweeping done, nor anything else to raise a dust, just before milking. The milker's hands should be carefully washed and dried, not moistened with milk, as is sometimes done. Milk pails and cans should be washed, as stated above, and always dried upside down, remaining in this position until used. Pails with a small opening partially protected by a hood should be used to lessen the contamination with dirt from the cows during milking. All sieves and straining cloths should be sterilised before each using. Milk should be bottled at the dairy, and so transported. Every time milk is handled, poured from one vessel into another, or in any way manipulated, the danger of contamination is increased.

As to temperature, no point in the care of milk is more important than the rapid first cooling; as soon as possible after being drawn it should be cooled to at least 50° F. Unless the milk is taken at once to a milk-house and some of the special forms of cooling apparatus employed, the cans should be immersed in spring water having a tempera-



ture below 50° F. or in ice-water, and remain at least one hour. If a temperature of 50° F. is maintained during transportation, which is quite possible if cans and bottles are properly iced, and during subsequent storage, the growth of bacteria may be so retarded that milk may be a safe food even when forty-eight hours old. If the temperature is not kept as low as 50° F. this result can not be depended upon, and with every degree above that point the increase in bacterial growth is very marked. Since the number of bacteria increases so rapidly with the age of the milk after the first twenty-four hours, it is of the utmost importance that milk be shipped as quickly as possible after it is collected.

A provision of the Sanitary Code of New York City requires that no milk shall be sold having a temperature above 50° F. This ordinance has done more than anything else to improve the milk supply of the city, especially to insure proper icing during transportation.

The desirable results indicated above are to be secured, in the first place, by educating the public to appreciate, and dealers to produce, a better and cleaner milk; secondly, by giving to the health authorities of city and state greater power than heretofore in the matter of milk inspection; thirdly, by the formation of milk commissions, through which the physicians of a town or city may co-operate to secure adequate supervision of at least a portion of the milk supply.

**Composition of Cow's Milk.**—Except in the percentage of fat, the composition of mixed or herd milk varies but little, whatever the breed. The fat is lowest in the Holsteins, and highest in the Jerseys.

*Composition of Cow's Milk.<sup>1</sup>*

|              | Jerseys. | Holsteins. | Average good herd milk. |
|--------------|----------|------------|-------------------------|
| Fat.....     | 5.61     | 3.46       | 4.00                    |
| Sugar.....   | 5.15     | 4.84       | 4.50                    |
| Protein..... | 3.91     | 3.39       | 3.50                    |
| Salts.....   | 0.74     | 0.74       | 0.75                    |
| Water.....   | 84.59    | 87.57      | 87.25                   |
| Total.....   | 100.00   | 100.00     | 100.00                  |

<sup>1</sup> In the table the figures for Jersey and Holstein herds are the averages given by the New York State Experiment Station. The legal requirements in New York and most of the States are, fat, 3 per cent; total solids, 12 per cent.

The figures given for herd milk are a little lower for the protein and a little higher for the sugar than in the older analyses. It is with milk of such composition that the average physician has to do in infant feeding. In a poor milk the only important difference to be considered is that the fat is from 0.5 to 1 per cent lower than the averages given. In a rich Jersey milk the chief difference is that the fat is 1 to 1.5 per cent higher than the averages; there is also an increase in the protein

and sugar which is less important, but should not be ignored. The variations in the fat content of milk are those which are of most practical importance to the physician. As to the relative advantages of the different breeds for this purpose, the difference does not seem great, provided all are equally healthy. Jerseys and all highly bred animals are more prone to disease and minor disturbances than the hardier common breeds.

**The Examination of Cow's Milk.**—The application of heat often causes coagulation in milk which is near the souring point, and also in colostrum milk. Both are unfit for use. The normal reaction of cow's milk is amphoteric or slightly acid. If strongly acid it should not be used; if alkaline, it is pretty certain that something has been added to it.

The *specific gravity* is from 1.028 to 1.033. If the milk has been falsified by the removal of cream, the specific gravity is raised; if adulterated by the addition of water, the specific gravity is lowered.

The best of all ready methods of determining fat are the Leffman and Beam and the Babcock tests.<sup>1</sup> By both, the fat is brought to the surface by the centrifuge after the addition of sulphuric acid and other reagents. These tests are similar, but differ in the reagents used. When carefully made they are very accurate. For institutions such an apparatus for determining the fat is indispensable; and the composition of the milk and the cream used can be determined each day.

The cream-gauge may be used as for woman's milk, the 100 c.c. size to be preferred; but it is not to be relied upon unless the milk is put into the cylinder soon after it is drawn and cooled rapidly by being placed in ice-water. Under these conditions, if the reading is made at the end of eight or ten hours the percentage of cream to that of fat is about three to one. If the milk has been first cooled and afterward handled two or three times before the test is made, the cream does not rise regularly, and the above ratio is not maintained.

A *microscopical examination* of milk is of considerable importance, and in cases where the character of the supply is questionable it may give valuable information. Both the cream and the sediment should be examined. Not much can be learned from a study of the fat globules, but among them may be found colostrum corpuscles, which are usually present for nearly a week after calving. The sediment is best studied after centrifuging. It should be examined for pus cells and blood, and stained for bacteria. A few leucocytes are almost invariably found in normal milk. Stokes and Wegefarth consider that an average of more than five in each field examined with an oil-immersion lens should be

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<sup>1</sup> The apparatus can be obtained of D. H. Burrell & Co., Little Falls, N. Y. The one sold as the "Facile Junior" may be used for woman's milk, urine, and other fluids as well as for cow's milk, and is very convenient for physicians' use. Price, \$10.

regarded as abnormal, and such milk excluded. The most frequent source of pus cells in numbers is inflammation of the udder. Pus cells may be associated with a stringy mucus. Blood may also result from inflammation of the udder, sometimes from traumatism.

When pus cells are present the specimen should be examined for bacteria. Any of the ordinary pyogenic cocci may be found. Streptococci were found by Eastes in 75 per cent of 186 specimens examined, although in most of these the number was so small that no symptoms were produced. He cites one instance where symptoms were caused. Woodward has reported a striking example where a family of five children were all made seriously ill with vomiting and collapse after taking milk which was found by him to contain large numbers of streptococci. The only certain way of demonstrating the presence of tubercle bacilli is by animal inoculation.

Whenever pus cells, blood, or streptococci are at all numerous, the milk should be regarded as unfit for food and a thorough inspection of the herd should be made.

**The Differences between Cow's Milk and Woman's Milk.**—Cow's milk is more opaque than woman's milk, although the latter may contain more fat. This opacity is due to the large proportion of calcium phosphate with which the casein is combined.

The *reaction* of cow's milk soon after it is drawn becomes acid. It is almost invariably so found unless some alkali has been added. Woman's milk is distinctly less acid.

The *specific gravity* and total solids in the two milks are about the same.

The *sugar* of both cow's and woman's milk is lactose in solution. The difference in amount is considerable. Cow's milk usually has 4.5 per cent, while woman's milk usually has from 6 to 7 per cent.

The greater part of the *fat* of cow's milk is neutral fat, as in woman's milk; cow's milk, however, contains a much larger proportion of the lower or volatile fatty acids than does woman's milk. Woman's milk on the contrary contains more oleic acid.

The *protein* of cow's milk is two and a half times as abundant as that of woman's milk, and it shows marked differences in character. Our knowledge of the protein both of cow's milk and woman's milk is still imperfect. The separation of the different forms of protein is difficult, and for this reason chemists are by no means agreed as to the proportions in which the different ones are present. It is well established that in woman's milk the soluble proteins—lactalbumin, etc., are in excess of the insoluble casein, Koenig giving the proportion as 5 to 4; in cow's milk, on the other hand, the proportion of the soluble protein is much smaller than the insoluble, the latest writers giving the proportion as 1 to 3.

The casein<sup>1</sup> of cow's milk is readily coagulated by rennet and acids. The curd formed by the gastric juice is tough and firm and is more slowly dissolved by the action of the digestive fluids. The casein of woman's milk is not regularly coagulated by rennet, and only slightly and with difficulty by acids. The curd formed by the gastric juice is loose and flocculent, and is readily and completely dissolved.

The *inorganic salts* in cow's milk are a little more than three times as abundant as in woman's milk. The most important differences in the composition of these salts are shown in the following analyses:

*Ash in 100 Parts of Milk (Bunge).*

|                           | Cow's. | Woman's. |
|---------------------------|--------|----------|
| Potassium oxide . . . . . | .0703  | .1760    |
| Sodium oxide . . . . .    | .0257  | .1110    |
| Calcium oxide . . . . .   | .0343  | .1590    |
| Magnesium oxide . . . . . | .0065  | .0210    |
| Ferric oxide . . . . .    | .0006  | .0003    |
| Phosphoric acid . . . . . | .0469  | .1970    |
| Chlorine . . . . .        | .0445  | .1690    |
| Total . . . . .           | .2288  | .7970    |

Cow's milk contains relatively a much larger amount of calcium phosphate and a smaller amount of potassium salts and of iron. The ash does not accurately represent the mineral constituents of milk. About 8 per cent of the phosphoric acid of the ash, according to Richmond, is derived from the phosphorus of the casein; while the traces of sulphuric and carbonic acid found are not true mineral constituents of milk. Most of the more recent analyses show the presence of citric acid in both woman's and cow's milk.

Cow's milk always contains a large number of bacteria, which increase in proportion to the age of the milk; woman's milk is either sterile or contains but a few cocci from the milk ducts.

**Cream.**—A great misapprehension exists as to its composition. It is often spoken of as if it were entirely different from milk. It should rather be regarded as milk which contains an excess of fat.

Cream was formerly obtained by skimming—the gravity process—at present, almost entirely by the use of a centrifugal machine known as a separator. The latter process has the advantage in point of time, as

<sup>1</sup> By Haliburton and some other chemists the term *caseinogen* is given to this protein as it exists in milk. When this is acted upon by rennet it splits up into two substances: One, the firm, insoluble coagulum to which only the term *casein* is applied; the other, a soluble protein which is known as *whey-protein*; this is present in but small amount. Those who use the term casein to designate the protein as it exists in milk refer to the curd formed by the action of rennet in the stomach as *paracasein*.

centrifugal cream can be put upon the market from twenty-four to thirty-six hours earlier than gravity cream.

The following table gives the composition of an average milk and of centrifugal cream of different densities removed from the same milk:

|              | Whole milk. | CREAM. |       |       |       |       |
|--------------|-------------|--------|-------|-------|-------|-------|
|              |             | I.     | II.   | III.  | IV.   | V.    |
| Fat.....     | 4.00        | 8.00   | 12.00 | 16.00 | 20.00 | 40.00 |
| Sugar.....   | 4.50        | 4.50   | 4.20  | 4.05  | 3.90  | 3.00  |
| Protein..... | 3.50        | 3.40   | 3.30  | 3.20  | 3.05  | 2.20  |
| Salts.....   | 0.75        | 0.70   | 0.65  | 0.60  | 0.55  | 0.45  |

The percentages of protein and sugar in the 8- and 12-per-cent cream are but little lower than in milk; in the very rich creams they are reduced by about one-third.

It is unfortunate that no general standard exists as to what shall be sold as cream. In New York State the law provides that cream shall contain at least 18 per cent fat. Very rich, centrifugal cream has from 35 to 40 per cent fat; the usual centrifugal cream has about 18 to 20 per cent. Gravity cream has generally from 16 to 20 per cent fat. It is possible to obtain from milk laboratories cream of any desired fat percentage.

None of the methods for determining the fat in milk is applicable to cream, except the Babcock test.

**Methods of Obtaining Milk Containing Various Proportions of Fat—Top-Milk, Skimmed Milk.**—To secure a milk for infant feeding which is fresh and at the same time one which contains a larger proportion of fat than does whole milk, the practice has come into vogue of using from milk purchased and delivered in bottles, only a certain number of ounces removed from the top. To this the term "top-milk" has been given. Different percentages of fat, which are sufficiently accurate for practical purposes, may be obtained by varying the amount removed. Top-milk and thin cream are practically identical in composition. If cow's milk is put into bottles soon after it is drawn and rapidly cooled, it will be found that after four hours the upper fourth will contain nearly all the fat that will rise as cream, and the top-milk may then be removed. It is therefore unnecessary to allow the milk to stand for a longer time. Milk bottled at dairies and then transported should be allowed to stand after it is received for at least two hours before removing the top-milk. This may be done with a siphon, spoon, or a small special dipper; pouring off is not so accurate.

Skimmed milk, or milk which contains a smaller proportion of fat than does whole milk, may be obtained from bottled milk by removing and rejecting a certain number of ounces from the top of the quart bottle and

using only the remainder. The amount of cream removed will depend upon the proportion of fat desired in the skimmed milk.

It is unnecessary in practice to have a top-milk which contains more than 7 per cent fat; while it is desirable at times to obtain milk which is practically fat-free. These two extremes and all intermediate proportions of fat may easily be obtained from bottled milk with approximate accuracy by the method given below. The results will of course not be the same with all milks, but will vary considerably according as the supply is from a good herd of selected cattle of mixed breeds (average 4 per cent fat), or from a Jersey or Alderney herd. It is therefore necessary for the physician to know with which one of these he is dealing, if the milk is to be used for infant feeding.

| If the original milk contains 4 per cent fat; |   |    |   | If it contains 5 per cent fat; |   |                          |   |
|---|---|----|---|--------------------------------|---|--------------------------|---|
| To obtain 7% fat, use upper 16 oz             |   |    |   | upper 20 oz. from 1 quart.     |   |                          |   |
| "   | " | 6% | " | "                              | " | 20                       | " |
| "   | " | 5% | " | "                              | " | 24                       | " |
| "   | " | 4% | " | "                              | " | all                      | " |
|   |   |    |   | remainder after skimming       |   |                          |   |
|   |   |    |   | off 2 oz.                      |   |                          |   |
| "   | " | 3% | " | "                              | " | remainder after skimming | " |
|   |   |    |   | off 3 oz.                      |   |                          |   |
| "   | " | 2% | " | "                              | " | 4                        | " |
|   |   |    |   | remainder after skimming       |   |                          |   |
|   |   |    |   | off 5 oz.                      |   |                          |   |
| "   | " | 1% | " | "                              | " | 8                        | " |
|   |   |    |   | remainder after skimming       |   |                          |   |
|   |   |    |   | off 8 oz.                      |   |                          |   |

Fat-free milk can be obtained only by the removal of the cream by a separator.

If the Jersey milk contains, as it often does,  $5\frac{1}{2}$  per cent of fat, 24 ounces should be removed from a quart bottle to secure a 7-per-cent milk; 28 ounces to secure a 6-per-cent milk; and 3, 5, 8, and 10 ounces respectively to obtain a skimmed milk which has 4, 3, 2, and 1 per cent of fat.

The physician should make or have made with the Babcock apparatus several fat tests of a given milk supply in order to obtain a basis upon which to make his calculations. In general it is wise for one who has much to do with infant feeding to have his patients take milk from the same supply to secure uniformity in his results.

In or near large cities it is possible to obtain from milk laboratories milk with any desired percentage of fat. This of course greatly simplifies the whole matter. How top-milk and skimmed milk of different percentages are used will be considered in the chapter on Infant Feeding.

**Milk Sterilisation.**—The term *sterilisation* is widely and rather loosely used to signify the heating of milk for the destruction of germs. It should, however, be borne in mind that none of the methods commonly employed renders milk sterile in the bacteriological sense of the word. What is accomplished is the destruction of such pathogenic germs as may be present, and from 95 to 99 per cent of the other bacteria, so

as to retard for a considerable time the ordinary fermentative changes. The preservation of milk for infant feeding, by boiling it in small bottles, was advocated by Jacobi many years ago.

The advantages of sterilising milk are obvious. When we consider the enormous number of bacteria present in cow's milk with the usual methods of handling, and that none of these, so far as is now known, are advantageous, but that they are frequently the cause of disease, it is not strange that after its introduction by Soxhlet in 1886 the practice of heating milk used for infant feeding was rapidly adopted all over the world. Following him, the earlier experiments in sterilisation were made at 212° F., usually continued for an hour and a half, and this temperature is still largely employed on the Continent of Europe. Even this does not render milk safe for very long. Spores are not destroyed, and at ordinary room temperatures spore-bearing bacteria may soon develop in such numbers as to make the milk dangerous. Since some of these bacteria act upon the milk-protein and not upon the sugar, such milk may not be sour, and hence its danger may not be recognised.

There are disadvantages in heating milk. The change in taste and the constipating effects of sterilised milk were soon noticed; other alterations were not so evident and have more recently come to be appreciated, although many of these are not yet fully explained. Some of the lactose is converted into caramel, causing a slight change in colour; the lactalbumin is partially coagulated, this beginning at 160° F. (70° C.); the casein is rendered less coagulable by rennet, and appears to be acted upon more slowly both by pepsin and trypsin; Rettger has shown that when milk is heated above 185° F. (85° C.) a volatile sulphide is liberated, conclusive evidence of a change in the protein; the organic phosphorus is changed into an inorganic phosphate; the citric acid is partially precipitated as calcium citrate, and some lime salts, which are usually soluble, are converted into insoluble compounds. Some changes also occur in the fat. Moreover, certain natural ferments in fresh milk, believed to be of value in digestion, are destroyed by heat.

Many of these changes are but imperfectly understood, and some of them are doubtless without any injurious effect upon nutrition. There is, however, one important clinical reason for believing that the nutritive properties of milk are impaired by heating to 212° F.—viz., the occurrence of scurvy in infants who are fed upon such milk for a long time. Of 379 cases of infantile scurvy brought together in the Report of the American Pædiatric Society in 1898, sterilised milk was the previous diet in 107. At least a score of such cases have come under my own notice. Again and again cases of scurvy have been cured by simply ceasing to sterilise the milk.

**Heating at Lower Temperatures—Pasteurising Milk.**—To obviate the disadvantages above referred to, the practice has come largely into use

in America of employing much lower temperatures for milk sterilisation, owing chiefly to the work of Freeman (New York) and Russell (Wisconsin).

At first 167° F. (75° C.) was used; subsequently, however, a lower temperature was found sufficient, and 150° to 155° F. (65° to 68° C.) are the temperatures which are now generally employed. These temperatures are maintained from twenty to thirty minutes. This is sufficient to kill the bacilli of tuberculosis, diphtheria, and typhoid fever, and from 98 to 99.8 per cent of all other bacteria in milk. Nearly all of the objectionable changes produced in sterilised milk are avoided when the temperature is raised only to 150° F. (65° C.), while it accomplishes the purpose for which milk is heated. The advantages of this form of heating are therefore obvious. But spores are not destroyed, and such milk requires special handling. It should always be rapidly cooled and kept at a low temperature. Pasteurised milk should be used within a few hours after heating; no attempt should be made to keep it more than twenty-four hours, even upon ice.<sup>1</sup>

**Pasteurisation vs. Sterilisation.**—From what has already been said it would appear that the argument is altogether in favour of pasteurisation. The lowest temperature and the shortest time that will surely destroy the objectionable bacteria in milk would seem to merit general

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<sup>1</sup> Quite distinct from the process just described is that known as *commercial pasteurisation*. In this, by passing milk through hot pipes, it is heated to temperatures ranging from 140° F. for several minutes to 160° F. for a very brief period, usually for 5 to 30 seconds. Such heating destroys from 90 to 99 per cent of the bacteria ordinarily found in milk. According to the experiments made in the laboratory of the New York Health Department, a temperature of 160° F. maintained for 30 seconds under usual conditions kills typhoid, diphtheria, and colon bacilli. In a small percentage of experiments about 1 in 100,000 of these bacteria withstood this exposure.

By this treatment (160° F. for 30 seconds) the great majority of tubercle bacilli, which are the most resistant of the bacteria exciting disease that are found in milk, are either killed or so injured that they can not infect. On the average about one-tenth of 1 per cent survive; 160° F. for one minute usually kills all.

The pasteurised milk of commerce which is extensively sold in many large cities is chiefly milk that has been heated for from 5 to 30 seconds in the manner described. The destruction of pathogenic organisms is a great advantage. The killing of the bacteria which produce the souring of milk makes it possible to keep milk in warm weather a much longer time before souring occurs. It is therefore a great advantage to the dealer, and he is likely to depend upon it rather than upon adequate icing and cleanliness in handling his milk. There are some serious objections to commercial pasteurisation. Milk so heated should be quickly cooled, should be received into sterilised vessels and kept at a low temperature (below 50° F.). If these precautions are not taken bacteria develop rapidly and the milk may after 24 hours be more dangerous than if it had not been heated at all; since, unlike raw milk, it does not usually sour and reveal its contaminated condition. Commercial pasteurisation should be permitted only under the most careful restrictions, and the can or bottle containing pasteurised milk should indicate the degree and time of heating.



adoption. Pasteurisation, however, requires considerable care, intelligence, and special apparatus; if not properly done it may be worse than nothing. Moreover, pasteurised milk can not, in very hot weather, be kept without ice as long as it may be necessary to keep milk. Sterilisation at  $212^{\circ}$  F. ( $100^{\circ}$  C.) is much simpler; it may be done with many simple and inexpensive forms of apparatus or even without any special apparatus. Where no ice is available, it is certainly safer in hot weather than pasteurisation. Among the poor of our large cities, in summer, heating to  $212^{\circ}$  for an hour is to be advised as the most satisfactory, and indeed the only efficient, method of sterilisation. It should not be forgotten that the use of such milk as the sole diet for a long time is attended with a certain amount of risk; and one should always be on the watch for the soreness of the legs and the spongy gums that indicate the beginning of scurvy, as well as for the more general symptoms of malnutrition. Heating to  $212^{\circ}$  F. on two successive days is also to be recommended where milk must be kept for one or two weeks, as upon ocean journeys.

**Methods of Heating Milk.**—Milk should be *sterilised* preferably in small bottles, each one of which contains a sufficient quantity for one feeding. These bottles may be plugged with cotton or corks, or special stoppers may be used. Soxhlet's apparatus may be employed, or Arnold's, or any one of a half dozen others sold in the shops. All that is really necessary is to expose the bottles on all sides to live steam in a closed vessel. It can be done effectively in any tin vessel which has a closely fitting cover and a perforated bottom, and which can be placed



FIG. 25.—FREEMAN'S PASTEURISER.  
A, Bottles in position for heating; B, method of cooling.

over a pot of boiling water. Sterilisation at  $212^{\circ}$  F. is usually continued for one hour. The bottles should then be cooled in water as quickly as possible and placed upon ice or in the coolest place available.

A simple apparatus for *pasteurising* milk has been devised by Freeman (Fig. 25). In this the temperature is raised to  $155^{\circ}$  F. ( $68^{\circ}$  C.)

by hot water.<sup>1</sup> Another useful form of apparatus is that of the Walker-Gordon Laboratory Company, which contains a thermometer so that any desired temperature can be secured. An essential step in pasteurising milk is rapid cooling. After forty-five minutes the bottles should be removed from the pasteuriser and placed in tepid water and afterward in ice-water, where they should remain half an hour before being placed in the cold room or ice chest.

**Limitations of Milk Sterilisation.**—While pasteurising or sterilising milk kills nearly all the living organisms, it destroys few of the spores, and probably but a small proportion, if any, of the toxins. Before sterilisation milk may contain the products of bacterial growth in such quantity and of such a character as to render it unfit for food. Again, the fewer the spores and spore-bearing bacteria which the milk contains, the more effective the sterilisation. The cleaner the milk the better will be the result.

Sterilised milk requires the same modification for infant feeding as raw milk. There is no evidence to show that its digestibility is enhanced by the process of heating.

The sterilisation of milk is useful, first, for the destruction of pathogenic germs, particularly typhoid and tubercle bacilli; secondly, for the destruction of the bacteria causing fermentation, thus enabling one to feed with safety milk in which, though it may be forty-eight hours old, no important fermentative changes have occurred. As a therapeutic measure sterilised milk is useful in various forms of gastric or intestinal infection such as typhoid fever, dysentery, diarrhoea, etc. In certain of these conditions no milk is admissible; at other times sterilised milk may be given when raw milk would be harmful.

**Shall all Milk used for Infant Feeding be Sterilised?**—In summer only the cleanest milk which has been handled in the best manner can safely be used without heating. In winter, the heating of milk is not necessary, provided the source of supply is known to be good. So long as

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<sup>1</sup> Freeman's apparatus is used as follows: The pail is filled to the groove with water, which is then raised to the boiling point. The bottles of milk are dropped into their places in the cylindrical cups, sufficient water being poured into each cup to surround the bottle, this water acting as the conductor of heat. The pail is now removed from the stove and placed upon a board or other non-conductor, and the receptacle containing the bottles of milk is set inside and the cover replaced. The volumes of milk and water have been so calculated that in ten minutes they are both at a temperature of 155° F. The water contains heat enough to maintain this, with very slight variations, for twenty minutes. In half an hour the bottles of milk are removed and cooled rapidly by being placed in a water-bath, the water being changed once or twice; or, better, by setting the pail in a sink and allowing the cold water to run from a faucet through a piece of rubber pipe into the pail, overflowing into the sink. This rapid cooling is very important. The bottles are then put in the refrigerator. This apparatus may be obtained from James Dougherty, 411 West Fifty-ninth Street, New York.

routine use it should be diluted with from eight to twelve parts of water, and sugar added.

**Dried Milk.**—Dried milk sold under various names has recently been put upon the market. It is prepared either from whole milk or from skimmed milk. The process of manufacture most extensively employed is that of spraying the milk upon hot revolving cylinders by which means the water is driven off almost instantaneously. Dried milk is a sterile white powder and in sealed cans keeps indefinitely. When eight parts by volume of water are added (one level teaspoonful to the ounce) it approximates in composition the original milk. It may be further modified if desired. Its application is similar to that of condensed milk over which it presents obvious advantages in travelling; it is open to the same objections as a permanent food, and should not be advised when fresh milk can be obtained.

**Buttermilk and Other Forms of Fermented Milk.**—Various forms of fermented milk are in use which differ according to the milk used and the process followed. They resemble each other in that the fermentation is excited by some of the varieties of lactic acid organisms, in some cases with the addition of yeast, which ferment a portion of the milk sugar. The ordinary buttermilk of commerce is sometimes made from sweet, but usually from sour cream. If from the latter, it resembles the fermented milks in that it contains little or no fat but a certain amount of lactic acid, the result of fermentation. It differs from them in that the fermentation in buttermilk is due to a great variety of lactic acid organisms; besides, it contains many other forms of bacteria than those concerned in the process of fermentation. Buttermilk should be made with care or it may be grossly contaminated. It, therefore, varies greatly in taste and considerably in composition at different times and under different conditions.

*Buttermilk (Vieth).*

|                       |        |
|-----------------------|--------|
| Fat .....             | 0.50   |
| Milk sugar .....      | 4.06   |
| Lactic acid .....     | 0.80   |
| Protein .....         | 3.60   |
| Inorganic salts ..... | 0.75   |
| Water .....           | 90.29  |
|                       | <hr/>  |
|                       | 100.00 |

When used as an infant food it is usually sterilised by boiling so that the living organisms are not given. Its low sugar content is overcome by the addition of milk sugar or cane sugar, sometimes also by barley flour or other farinaceous food, in any proportion desired. A formula much used in Europe is: buttermilk, one quart; barley flour, two even tablespoonfuls; water, four ounces. Cook slowly, constantly stirring, for twenty minutes; then add two teaspoonfuls of cane sugar. The advantages of buttermilk as an infant food are chiefly due to its

low fat content and the small amount of lactic acid which it contains. Its cheapness is an important consideration and makes it available for the very poor.

Other fermented milks, sometimes called buttermilk, are known also as *lactic acid milk*, *lactobacilline*, *lactobacillary milk*, *lactone buttermilk*, etc. They are sometimes made from whole milk but chiefly from skimmed milk. This is usually first sterilised and then the ferment added in the form of tablet, mixture or culture from some previously fermented milk. The ferment consists of different varieties of lactic acid organisms; the one most frequently employed is known as the *Bulgaricus*. The product differs according to the exact varieties or combinations used, also according to the temperature maintained and the duration of the fermentation. A temperature of 80° to 85° F. is usually employed and this is continued from twelve to twenty-four hours according to the degree of acidity desired. The milk is then bottled and put on ice, where a slight change continues, although the milk alters but little for several days. The taste is rather pleasant unless the acidity is too pronounced. The product always contains a considerable amount of lactic acid; it should not contain alcohol or acetic acid. These fermented milks are sometimes used in acute disease, but chiefly in chronic intestinal conditions. They are not adapted to continuous use in infant feeding.

*Kumyss* has been made by the Tartars for centuries from mare's milk. It is made in this country from cow's milk, sometimes skimmed, but usually from the whole milk. The fermentation is generally started with yeast and is continued in corked bottles usually for several days, with frequent agitation. *Kumyss* contains carbon dioxide, lactic acid, alcohol and traces of butyric and acetic acid. The acidity and the taste depend upon the duration of the process.

*Zoolak* or *matzoon* is made from whole milk which is first sterilised and then has added to it a ferment which contains some form of yeast. It differs from *kumyss* chiefly in that the process is carried on in open vessels and the carbon dioxide allowed to escape. It is a thick smooth liquid and has a taste resembling that of sour cream.

Both *kumyss* and *zoolak* are better adapted for use with older children than with infants; they are chiefly valuable in cases of chronic intestinal indigestion. For infants they should be diluted with water and often given with a spoon since they are too thick to go through the ordinary nipple.

*Protein Milk* (*Eiweiss Milch* of Finkelstein).—The object of this preparation is to secure a milk for infant feeding which is low in sugar, high in protein with a moderate amount of fat. It is made as follows: To one quart of whole milk is added half an ounce of rennet or enough to coagulate the casein. The whey is strained off through muslin, by suspending the curd for an hour. The curd is then rubbed through a

Beef extracts are not to be considered in any sense as foods. Kemmerich has shown that animals receiving nothing else died of starvation, and sooner even than when everything was withheld. According to Chittenden, they contain no nitrogen in the form of protein, but only in combination with the soluble extractives. They are stimulants, but as such are often useful.

Rare scraped beef is easily digested by most young children. There are many conditions in which other forms of protein are not well borne, where children even as young as twelve months appear to digest this beef-pulp without difficulty. It should be made from very rare or raw steak, finely scraped and well salted. A tablespoonful may be given at one feeding to a child of eighteen months. In nutrient properties this far exceeds the beef preparations in the market. The alleged danger of tapeworm from the use of rare scraped beef or beef juice is in this country so slight that it may be disregarded.

**Broths.**—Animal broths may be made from mutton, veal, chicken, or beef. A good formula for general use is the following: One pound of lean meat, one pint of water; stand for two hours, then cook over a slow fire for two hours down to half a pint. After it has cooled, skim off the fat and strain through a cloth. The composition of a broth so made is given by Cheadle as follows:

*Beef Broth.*

|                   |        |
|-------------------|--------|
| Protein .....     | 1.02   |
| Extractives ..... | 1.82   |
| Fat .....         | 0.00   |
| Salts .....       | 0.88   |
| Water .....       | 96.28  |
|                   | <hr/>  |
|                   | 100.00 |

From their composition it will be seen that broths are not very nutritious; they are, however, quite stimulating, and are at times useful, particularly where milk must be temporarily withheld. They are, however, not adapted to prolonged use alone. Broths which have been thickened with either barley or rice flour are useful for infants and older children.

**Albumin Water.**—This is prepared as follows: The white of one fresh egg is mixed with a pint of cold water, a little salt, and a teaspoonful of brandy added. It should be given cold. Albumin water is useful in a variety of conditions attended by gastric irritability. The nutritive value of this preparation, it should be borne in mind, is very small.

**CEREALS.**

**Barley Water.**—This may be made either from the grains or from the barley flour. When the grains are used, the following is the formula which I have been accustomed to employ: To two tablespoonfuls of pearl

barley, add one quart of water and a pinch of salt, and boil continuously for six hours, keeping the quantity up to a quart by the addition of water; strain through coarse muslin. It is an advantage to soak the barley for a few hours before cooking. The water in which it is soaked is not used. When cold this preparation makes a rather thin jelly. Its composition by analysis is as follows:

*Barley Water.*

|                       |        |
|-----------------------|--------|
| Starch .....          | 1.63   |
| Fat .....             | 0.05   |
| Protein .....         | 0.09   |
| Inorganic Salts ..... | 0.03   |
| Water .....           | 98.20  |
|                       | <hr/>  |
|                       | 100.00 |

Almost an identical product may be obtained in an easier way by using prepared barley flour, one even tablespoonful to each twelve ounces of water, and cooking for twenty minutes. A thicker jelly when desired can be made by using twice as much of the barley.

**Rice, Wheat, or Oatmeal Water, etc.**—These may be made in the same manner as the barley water, using the same proportions either of the flour or the grains. These are useful as additions to milk for healthy infants who have reached the age of five or six months; they may also be given in many cases of acute or chronic indigestion where milk must be omitted or given in small quantities. When there is a tendency to constipation oatmeal is preferred; when to looseness, barley, wheat, or rice water.

INFANT FOODS.

It is not possible, nor even desirable, for a physician to know all about the infant foods with which the market is flooded. He should, however, know at least that they are not perfect substitutes for breast-milk, that as permanent foods they are greatly inferior to properly modified cow's milk, and that they are capable of doing and have done much positive harm. Rickets and scurvy have so frequently followed their prolonged use, when given without the addition of fresh milk, and sometimes even when they have been given with it, that there can be no escaping the conclusion that they were the active cause. Their general use is condemned with practical unanimity by authorities on infant feeding. Yet by industrious and skilful advertising they are forced upon public attention, and are extensively used by the laity and even by the medical profession. They are expensive. They add little or nothing to our resources in infant dietetics; in fact, they tend to retard rather than advance our knowledge of this subject.

There are, however, a few occasions when some of these preparations

may be useful as temporary expedients or when nothing better can be obtained. They should be used only with a very definite knowledge of exactly what they do and what they do not contain. Their name is legion; but those most commonly employed in this country may be grouped as follows:

1. **The Milk Foods.**—Nestlé's food is perhaps the most widely known. The others closely resembling it in composition are the Anglo-Swiss, the Franco-Swiss, the American-Swiss, and Gerber's food. These foods are essentially sweetened condensed milk evaporated to dryness, with the addition of some form of flour which has been dextrinised; they all contain a considerable proportion of unchanged starch.

2. **The Liebig or Malted Foods.**—Mellin's food may be taken as a type of the class. Others which resemble it more or less closely are Liebig's, Horlick's malted milk, and cereal milk. Mellin's food is composed principally (80 per cent) of soluble carbohydrates. They are derived from malted wheat and barley flour, and are composed chiefly of a mixture of dextrin, dextrose, and maltose.

3. **The Farinaceous Foods.**—These are imperial granum, Ridge's food, Hubbell's prepared wheat, and Robinson's patent barley. The first consists of wheat flour previously prepared by baking, by which a small proportion of the starch—from one to six per cent—has been converted into sugar. In chemical composition these four foods are very similar, consisting mainly of unchanged starch which forms from seventy-five to eighty per cent of their solid constituents.

4. **Miscellaneous Foods.**—Under this head may be mentioned Carnrick's soluble food and Eskay's food. The composition of these is given in the following table:

*The Composition of Infant-Foods.<sup>1</sup>*

|  | Nestlé's food. | Mellin's food. | Eskay's food.      | Malted milk.       | Ridge's food. | Imperial granum. | Carnrick's food. |
|--|----------------|----------------|--------------------|--------------------|---------------|------------------|------------------|
|  | Per cent.      | Per cent.      | Per cent.          | Per cent.          | Per cent.     | Per cent.        | Per cent.        |
| Fat . . . . .                              | 5.50           | 0.24           | 1.16               | 8.78               | 1.11          | 1.04             | 7.45             |
| Protein . . . . .                          | 14.34          | 11.50          | 5.82               | 16.35              | 11.81         | 14.00            | 10.25            |
| Cane sugar . . . . .                       | 25.00          | .....          | .....              | .....              | .....         | .....            | .....            |
| Dextrose . . . . .                         | .....          | .....          | 53.46 <sup>2</sup> | .....              | 0.52          | 0.42             | .....            |
| Lactose (milk sugar) . . . . .             | 6.57           | .....          |                    | 49.15 <sup>3</sup> | .....         | .....            | .....            |
| Maltose . . . . .                          | 27.36          | 60.80          | .....              |                    | .....         | .....            | .....            |
| Dextrins . . . . .                         |                | 19.20          | 14.35              | 18.80              | 1.28          | 1.38             | .....            |
| Total Soluble carbohydrates . . . . .      | 58.93          | 80.00          | 67.81              | 67.95              | 1.80          | 1.80             | 27.08            |
| Insoluble carbohydrates (Starch) . . . . . | 15.39          | .....          | 21.21              | .....              | 76.21         | 73.54            | 37.37            |
| Inorganic salts . . . . .                  | 2.03           | 3.59           | 1.30               | 3.86               | 0.49          | 0.39             | 4.42             |
| Moisture . . . . .                         | 3.81           | 4.73           | 2.70               | 3.06               | 8.58          | 9.23             | 3.42             |

<sup>1</sup> With the exception of Nestlé's food and Carnrick's soluble food, these analyses were made for the author by E. E. Smith, Ph.D., M.D., of samples purchased in the open market.

<sup>2</sup> Chiefly lactose.

<sup>3</sup> Largely maltose.





# PLATE III.

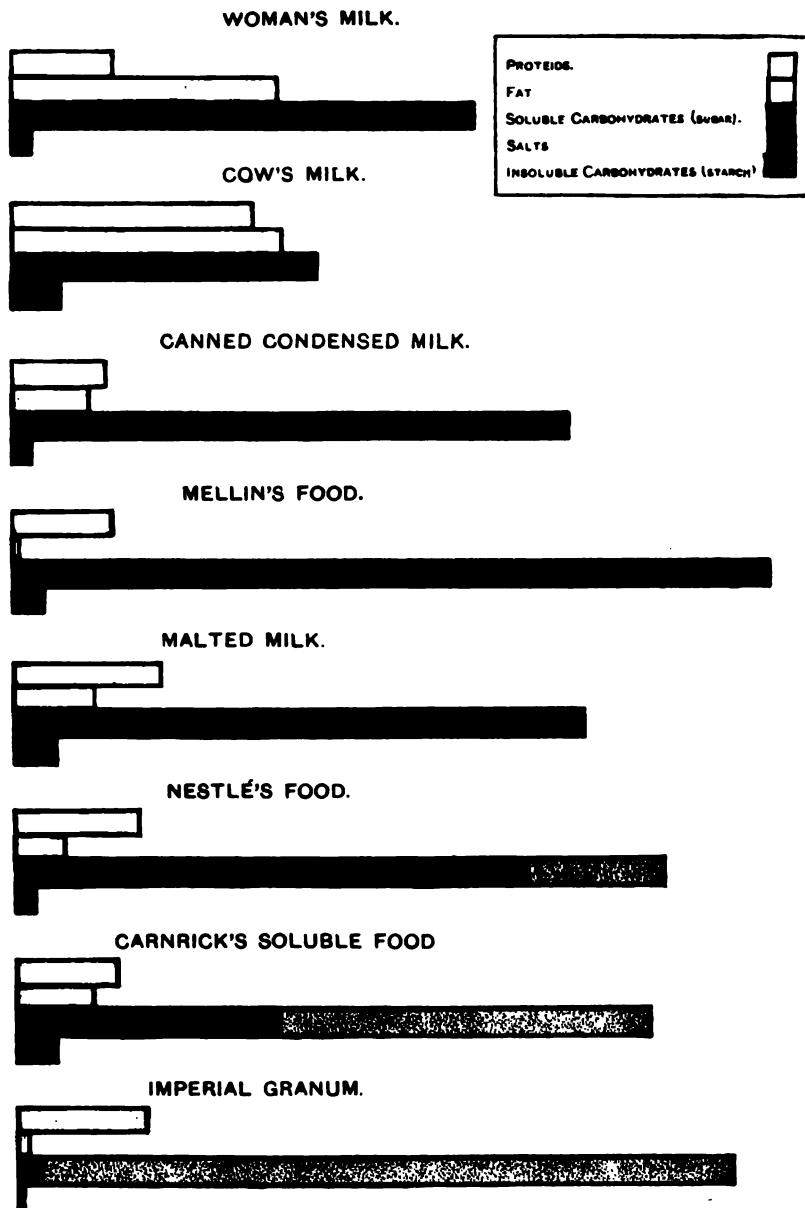


Chart showing the solid ingredients of various infant foods as compared with those of woman's milk.

A better idea of the composition of these foods can be obtained by a study of the accompanying chart (Plate III), which shows their solid constituents as compared with those of woman's milk. The essential features of the foods are seen at a glance—i. e., they are all composed principally of carbohydrates and are lacking in fat. Some of them contain a large proportion of unchanged starch. Furthermore, their protein, though often sufficient in amount, is chiefly vegetable, not animal protein. No one of them can be regarded in any sense as a proper substitute for breast-milk.

Some of these foods—Nestlé's and other milk foods, malted milk, cereal milk, and Carnrick's food, and even some of the farinaceous foods, like imperial granum—are advertised as substitutes for breast-milk and recommended for use alone. Others, such as Mellin's, Liebig's, and Eskay's foods, are intended to be used with milk. The use of any of the commercial foods alone is admissible only for short periods during derangements of digestion, when we wish to withhold for the time all fat and milk protein. Their prolonged use almost invariably produces some grave disorder of nutrition, most frequently rickets or scurvy. Those foods which require in their preparation the addition of milk are open to less serious objections, but are not necessary or even desirable. They should never be used with condensed milk. When added to fresh milk they may furnish the additional carbohydrates required by an infant fed upon a diluted cow's milk. In such a case they take the place of milk sugar or cane sugar in the milk modification. There is no proof to sustain the claim that they increase the digestibility of cow's milk. Farinaceous foods may be used as an addition to milk after the sixth or seventh month and during the second year.

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## CHAPTER III.

### *INFANT FEEDING.*

#### CHOICE OF METHODS OF FEEDING.

THE different methods of feeding which are available are:

1. Breast-feeding, either by the mother or by a wet-nurse.
2. Mixed feeding, or a combination of nursing and artificial feeding.
3. Artificial feeding exclusively.

In deciding by which one of these methods a child shall be fed, many circumstances must be taken into consideration: the vigour of the child, the health of the mother, and especially the surroundings, since these determine very largely the success or failure of any method employed.

**Maternal Nursing.**—This is the natural and the ideal method of infant feeding. Every mother should nurse her infant unless there are some very weighty reasons to the contrary. The physician should do all

in his power to encourage maternal nursing and to promote its success. He should explain to the mother how important breast-milk is for the child; that fully four-fifths of the deaths under one year are in infants who are artificially fed. He should also make clear the conditions by which alone successful nursing can be accomplished; viz., proper diet, regular habits of sleep and exercise, and a simple life, in so far as possible free from causes of nervous excitement, fatigue, over-work, or worry. Social engagements should be avoided. Nursing may be furthered by proper care of the nipples before delivery, and by attention to them during the early days of nursing to prevent fissures and mastitis, which so often interrupt successful nursing.

In spite of all efforts to the contrary, it is nevertheless a fact that the capacity for maternal nursing is steadily diminishing in this country, chiefly in the cities, but to a considerable degree in the rural districts as well. Among the well-to-do classes in New York and its suburbs, of those who have earnestly and intelligently attempted to nurse, less than 25 per cent, in my experience, have been able to continue satisfactorily for as long as six months. An average city mother who is able to nurse her child successfully for the entire first year is almost a phenomenon. Among the poorer classes in our cities a decline in nursing ability is also seen, although not yet to the same degree as in the higher social scale. These are facts that must be taken into account in deciding the question of feeding. While nothing is so good as good maternal nursing, no method of feeding gives much worse results than poor nursing. Among the classes of society where most of the maternal nursing is very poor, but where every facility can be afforded for the best artificial feeding, one should not be slow to adopt the latter in cases of doubt. Among the poor and ignorant, however, where artificial feeding can not be carried on with anything like the same chances of success, all possible efforts should be made to increase maternal nursing as the most effective means of reducing infant mortality.

*When Maternal Nursing should not be Attempted.*—(1) No mother who is the subject of tuberculosis in any form, whether latent or active, should nurse her infant; it can only hasten the progress of the disease in herself, while at the same time it exposes the infant to the danger of infection. (2) Nursing should seldom be allowed where serious complications have been connected with parturition, such as severe hæmorrhage, puerperal convulsions, nephritis, or puerperal septicæmia. (3) If the mother is epileptic. (4) If the mother is suffering from any serious chronic disease or is very delicate, since great harm may be done to her without any corresponding benefit to the child. (5) Where experience on two previous occasions under favourable conditions has shown her inability to nurse her child. (6) When no milk is secreted. With reference to the fourth and fifth conditions, an absolute opinion can not

always be given at the outset. As a rule, mothers are more likely to succeed in nursing first or second children than subsequent ones. My own statistics indicate that in general the capacity for lactation dimin-

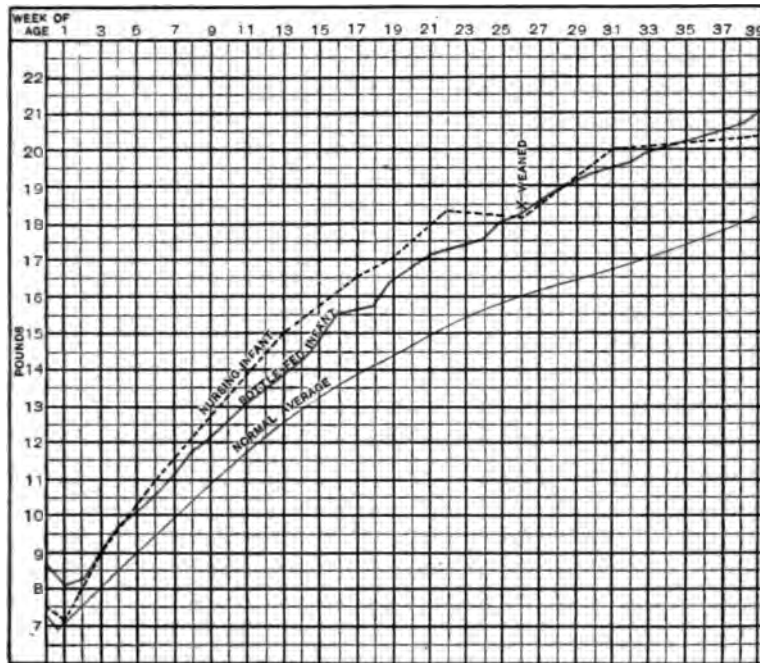


FIG. 26.—WEIGHT CURVE OF NURSING AND ARTIFICIAL FEEDING COMPARED. Both infants were strong, well nourished, and in good surroundings. The bottle-fed infant was never once put to the breast; fed from the milk laboratory. First formula: Fat 1 per cent, sugar 5 per cent, protein 0.5 per cent. At six weeks taking: Fat 3 per cent, sugar 7 per cent, protein 1.25 per cent. It will be observed that the nursing infant made more rapid progress during the first few weeks, while the bottle-fed infant more than made up for this between the fifth and ninth month, for weaning became necessary in the other child owing to the gradual failure of the mother's milk. The stationary weight was the result of this condition, and the irregular subsequent gain was incident to the change of food.

ishes with each successive pregnancy. My inclination as a result of increasing experience is not to allow nursing in either of these conditions, provided the means for proper artificial feeding can be commanded. The chances of success are so small and the difficulties are so increased by even a few weeks of bad nursing that I prefer not to put the child to the breast at all, even for the first two or three days. The breasts are bound up at once and kept bandaged. When one begins with healthy digestive organs the difficulties with artificial feeding are relatively few, and it is usually successful.

**Artificial Feeding vs. Wet-Nursing.**—When maternal nursing is impossible or undesirable, the milk of another woman would seem to be

the most natural and best substitute. While this is theoretically true, the practical obstacles are so many as to put wet-nursing out of the question as a general method of feeding. We have in America no peasant class like that of Europe to draw upon; and in the class which furnishes most of our wet-nurses the capacity to nurse has steadily diminished. The expense of a wet-nurse—twenty-five to thirty-five dollars a month in New York—the danger of transmitting contagious disease, and the difficulty of obtaining proper care for her own infant, are all very serious objections to wet-nursing. The recent advances in artificial feeding have placed it now on quite a different footing from that which it formerly occupied. While it is true that good breast-milk is unquestionably the best food, it is equally true that properly modified cow's milk is a far better food than the milk of many wet-nurses who are employed. These facts added to the constantly increasing difficulty of obtaining good ones have caused wet-nurses to be pretty generally discarded, even in our large cities, where formerly no other substitute for maternal nursing was considered.

There are, however, some conditions in which they are necessary, even indispensable. Some infants, usually those who have been badly started, can not be made to thrive upon any form of artificial feeding. There are also premature infants and other very delicate ones whose powers of assimilation are so feeble that they are reared under any circumstances only with the greatest difficulty, but whose chances of life are much increased by a good wet-nurse. Again, in young infants who have been suffering for some time from chronic indigestion and failing nutrition, the symptoms of acute inanition sometimes develop with great rapidity and severity. From such a condition, apparently hopeless, infants may sometimes be rescued by the timely assistance of a good wet-nurse.

The difficulties in the way of successful infant feeding in foundling asylums and other institutions for young infants are such that in them partial wet-nursing at least should be employed whenever possible.

**Mixed Feeding.**—Mixed feeding, or a combination of nursing and artificial feeding, may be employed whenever the supply of the nurse is insufficient. The use of one or two feedings a day from the bottle after the third or fourth month may do much to relieve the mother from the strain of nursing entirely without disturbing the infant's progress. During the later months more feedings may be introduced for the purpose of gradual weaning.

#### BREAST-FEEDING.

**Care of the Breasts during Lactation.**—For the safety of both mother and child it is essential that the most scrupulous attention be given to cleanliness. The nipples, and the breasts as well, should always be care-

fully washed after each nursing. Usually plain water is sufficient, or a weak boric-acid solution may be employed.

**Nursing during the First Days of Life.**—This is necessary, to accustom the child and the mother to the procedure, and to empty the breasts of the colostrum; it probably also promotes uterine contractions. All these results can be attained by putting the child to the breast on the first day once in six hours, on the second day once in four hours. The child gets from the breast only from four to six ounces a day during the first two days. Did it require more nourishment before the milk-flow is fully established, we may be sure that Nature would not have been so late with her supply. The common practice of administering to an infant a few hours old all sorts of decoctions, with the idea that because it cries it is suffering from colic, can not be too strongly condemned. A certain amount of crying is necessary. In exceptional circumstances, when an infant is unusually large and strong and cries excessively, it may be necessary to give food even on the first day; but this is not to be the rule. A little warm water should first be given; from two to four teaspoonfuls at a time are sufficient. If this does not satisfy the child, regular feeding should be begun on the second day. Should the milk be delayed beyond the second day, the child should be put to the breast at regular intervals, but only for two or three minutes, and then given the bottle thereafter if still hungry. It is important not to cease in our efforts to induce a secretion for several days longer, and the best of all means is the stimulation of the child's sucking.

**Nursing Habits.**—Good habits of nursing and sleep are almost as easily formed as bad ones, provided one begins at the outset. A vast deal of the wear and tear incident to the nursing period may be avoided if the child is trained to regular habits. Attention to these minor points often makes all the difference between successful and unsuccessful nursing. After the third day, ten nursings in the twenty-four hours are quite sufficient for the first weeks, and no more should be allowed. An infant at this age can usually be depended upon to take at least one long sleep of from four to five hours in the twenty-four. For the rest of the day the child should be awakened, if necessary, at the regular nursing time, and put to the breast; this plan being continued until nine o'clock at night. He should then be allowed to sleep as long as he will, and but two nursings given between this hour and seven in the morning. In the course of two or three weeks a healthy infant can usually be trained to nurse and sleep with almost perfect regularity, frequently, when a month old, going six hours regularly at night without feeding. A trained nurse of my acquaintance states that out of thirty-three infants of which she had the care from birth, thirty-one were trained without difficulty in the manner stated. Of course, success in training must rest almost entirely with the nurse; but the physician should at least appreciate the impor-

tance of proper training and lend it his support. So far as the child is concerned, regular habits of feeding and sleep, and regular evacuations from the bowels, which nearly always go with them, are most important factors in infant hygiene.

*Schedule for Breast-Feeding.*

| Age.                             | Number of nursings in 24 hours. | Interval during the day. | Night nursings between 9 P.M. and 7 A.M. |
|----------------------------------|---------------------------------|--------------------------|--|
| First day . . . . .              | 4                               | Hours.<br>6              | 1  |
| Second day . . . . .             | 6                               | 4                        | 1  |
| Third to twentieth day . . . . . | 10                              | 2                        | 2  |
| Third to ninth week . . . . .    | 8                               | 2½                       | 1  |
| Third to fifth month . . . . .   | 7                               | 3                        | 1  |
| After the fifth month . . . . .  | 6                               | 3                        | 0  |

Relieving the mother of night-nursing after the child is five months old is of the greatest value, and will often enable her to continue lactation, when otherwise it would be brought to an abrupt termination. On no account should the child be allowed to sleep upon the mother's breast, nor in the same bed with the mother. The temptation to frequent nursing is thus largely removed. No mere sentiment in regard to these matters should be allowed to interfere with the plain dictates of reason and experience.

**Symptoms of Unsuccessful Nursing during the Early Weeks.**—Attempts at maternal nursing so often result in failure, jeopardising the health, and even endangering the life of the child, that it becomes a matter of the greatest importance to decide this question of nursing aright, and as early as possible. On the one hand, one should not hastily wean a child on account of symptoms which may have no connection with the food, nor should one advise weaning when the indigestion from which the infant is suffering is due to causes which are temporary and remediable. On the other hand, nursing should not be continued simply because a conscientious mother desires it, when every indication points to failure. If artificial feeding is to be employed the difficulties are fewer when it is begun early than after the digestive organs have been deranged by several weeks of poor nursing. These cases form a very large group and present peculiar difficulties in practice. While a decision is being reached as to the ability of the mother to nurse, there is required close observation and a careful study of all the conditions, and even then the physician is liable to make mistakes in judgment the results of which may be serious.

The body-weight gives valuable information. The child does not gain or continues to lose after the usual initial loss of the first three or four days. Observations on the weight at least twice a week are

necessary, and in cases presenting special difficulties the weight should be taken daily.

At times there may be no vomiting, diarrhoea, or even severe colic, yet the child may fret and worry continually, sleep but little, and show a general discomfort. In other cases definite symptoms of gastric indigestion may be present, usually vomiting or frequent regurgitation of small amounts of undigested milk, later mixed with mucus; eructations of gas with or without vomiting may occur, and distention of the stomach with gas and gastric colic may follow.

More often the symptoms of indigestion are intestinal. Occasionally there is constipation, but as a rule the stools are frequent, thin and green, containing flaky masses of undigested milk, and, after a short time, mucus which is frequently in large amount. The odour of the discharges may be slightly sour or there may be none at all. At times there is much gas and the stools are sour and irritating. If constipation is present there is apt to be severe colic and abdominal distention. The almost uniform absence of any elevation of temperature in these cases points strongly against the existence of an intestinal infection, which is further indicated by the prompt recovery under appropriate treatment.

Before considering the case one of inadequate nursing, or simple indigestion in a nursing infant, one should be careful to exclude organic conditions in the child, particularly hypertrophic stenosis of the pylorus. The diagnosis of unsuccessful nursing should include the changes in the milk and if possible the causes of these changes.

As the first step one should endeavour to gain some idea as to the quantity of milk secreted. During the first week, particularly from the second to the fourth day, the temperature may be elevated quite apart from septic or inflammatory conditions or even evidences of indigestion. This is particularly seen where the breasts secrete almost nothing (see Inanition Fever). Often when the milk is very scanty something may be learned from the manner in which the child takes the breast. Where the milk is abundant, five or six minutes are often sufficient. If the milk is very scanty, an infant will frequently nurse half or three-quarters of an hour and then stop, more because he is exhausted than because he is satisfied. Sometimes, when the breasts are practically empty, the child will seize the nipple and nurse vigorously for a few moments, then drop it in apparent disgust and refuse to make any further efforts. The only satisfactory way of determining the quantity of milk secreted is to weigh the infant before and after each nursing. If the milk is merely scanty, but not otherwise abnormal, the infant does not gain, but shows no symptoms of indigestion, such as vomiting, colic, or undigested stools, and he frets and cries from hunger only.

An excessively rich milk is usually found under the following conditions: The mother is in good health, has large breasts which are full



and tense at nursing time. In most cases she is upon a very abundant diet, getting little or no exercise, and frequently taking some alcoholic beverage with the notion that because the child is not thriving the milk is poor. The child may be colicky, sleepless, and uncomfortable, may vomit, may have frequent stools containing much undigested food, and may be losing in weight. A similar condition is often seen when a wet-nurse makes a change from the simple life and habits of her own home to the more luxurious life and diet of the family to which she goes. The milk then has usually a high specific gravity, is high in fat and high in protein. The following analyses from Rotch illustrate the point: No. I shows milk of a healthy but under-fed wet-nurse two days before change of food; II, the milk of the same nurse after one month of rich food with very little exercise; III, milk of the same nurse, the food and exercise being regulated. The effect of the exercise and the change in diet is seen in a very marked reduction in the protein.

|              | I.        | II.       | III.      |
|--------------|-----------|-----------|-----------|
|              | Per cent. | Per cent. | Per cent. |
| Fat.....     | 0.72      | 5.44      | 5.50      |
| Sugar.....   | 6.75      | 6.25      | 6.60      |
| Protein..... | 2.53      | 4.61      | 2.90      |
| Salts.....   | 0.22      | 0.20      | 0.14      |

A scanty milk of a poor quality is most often seen when the mother is delicate or anæmic, or perhaps has had a difficult or complicated labour, and who besides is anxious and careworn. It is often with the greatest difficulty that one can secure the necessary half ounce required for examination. The milk is usually low in total solids and very low in fat. The specific gravity may be only 1.024 to 1.027, and the fat only one per cent or less.

A disturbed or disordered milk secretion is sometimes seen when the milk is scanty, often when it is very abundant. Like the group of cases just mentioned, this is frequently met with when the mother's general health is below the normal, but particularly is it influenced by her nervous condition. It is the highly nervous, emotional, worried woman whose milk we are now considering. During the first week or two the secretion may be excessive and then rapidly diminish; or, though the milk continues abundant, the infant shows no improvement. It is most frequently found on examination that the milk is low in fat (0.50 to 1 per cent), while it may be high in protein (1.75 to 3.50 per cent). The child's symptoms are usually those of intestinal indigestion—severe colic, flatulence, and frequent, green, undigested stools. Very similar symptoms are sometimes seen when the milk is high in fat.

*Management.*—The cause of the symptoms being in the food and not in the child, the futility of all medical treatment will be at once appar-

ent. He who expects to relieve the symptoms of indigestion by the use of digestive ferments, by giving something before the nursing to dilute the milk, or to check frequent intestinal discharges by opium or astringents, will be disappointed. Temporary benefit often follows a dose of castor oil, but unless the milk can be materially changed in composition no permanent improvement in the child is to be looked for. The question usually to be decided relates to the continuance of nursing. We have a choice of four courses: (1) To continue nursing, endeavouring to correct the milk through treatment of the mother; (2) to partly nurse and partly feed from the bottle; (3) to stop all nursing temporarily, pumping the breasts meanwhile to keep up the secretion while we attempt to improve its character; (4) to wean at once and entirely. In deciding which of these courses is to be adopted we must take into consideration the condition of the child, the severity and duration of his symptoms, the findings of the milk examination, and the condition of the mother.

While the analysis of the milk is of some value in determining the course to be pursued, and should, if possible, be made, it is of much less importance than the child's symptoms. We must be guided not by what the milk contains, but by how seriously it disagrees. The chemical examination may show the milk to be of normal average in the proportion of its different ingredients and yet the child be seriously upset by it; on the other hand, a child may be doing admirably upon a milk which shows proportions which differ very greatly from the normal average. The question always concerns the effect of the particular milk upon the particular child.

When the symptoms of indigestion are severe or have been prolonged it is usually a mistake to attempt to relieve the condition by simply substituting some other food for part of the nursings. This seldom leads to any material improvement in the symptoms, while it does confuse the result, since we can not now tell whether it is the breast or the bottle feeding which disagrees. A better plan is to stop nursing entirely for a time and try the bottle alone. If the symptoms are at once relieved the weaning should be permanent.

When symptoms point to a scanty milk, but of fair quality—i. e., infant not gaining but without any particular symptoms of indigestion—one is often able to overcome the difficulties and continue the nursing to advantage. Until a decided increase in the milk has occurred the child should have supplementary feedings from the bottle in sufficient number to insure his being properly nourished. Only one or two a day may be required, or it may be desirable to nurse and give the bottle alternately. If the latter plan is followed, both breasts should be given at each nursing period for the stimulating effect upon the secretion.

In the treatment of the mother the first thing is to secure for her an undisturbed rest at night. If possible, she should be entirely relieved of

the care of the infant at this time, and if feeding is necessary the bottle should be given. She should have a certain amount of fresh air every day, driving if possible, or walking as soon as she is able to take more active exercise. Gentle massage of the breasts is often useful in stimulating secretion. It should be done with care and with every precaution against infection, and may be repeated two or three times a day for ten minutes. The diet should be abundant, with a large allowance of milk and meat, especially beef. If there is anæmia, iron should be given. Every means should be taken to improve the general nutrition, for whatever benefits this improves the milk. If the conditions present are incident to the confinement or the convalescence, the prognosis is good; and in the course of a week or two very marked improvement may be evident, and lactation may be successfully continued. If, however, the conditions depend upon constitutional debility, the prognosis is much worse. Temporary improvement may take place, but it soon becomes evident that the nursing is a failure.

When the symptoms are found to be associated with an over-rich milk the prospects for continuing nursing are much better than when the milk is poor. Unless the infant's digestion is very feeble or has been seriously upset either with vomiting or diarrhœa, one can usually so alter the milk by treating the mother as to make it possible to keep the baby at the breast. Alcohol should be prohibited; the diet, especially the amount of solid food, should be reduced, and the mother required to take daily exercise in the open air, particularly by walking. The intervals between nursings should be lengthened, usually to three hours. In many cases there is an advantage in diluting the milk by allowing the child to take water before putting it to the breast. The improvement following such a change in regimen is often immediate, and with increasing age and weight the child gradually becomes accustomed to and is able to digest the rich milk. If, however, the child's symptoms of indigestion are of an aggravated type, whether gastric or intestinal, it will be necessary, even though the weight is increasing normally, to stop nursing entirely for a time. The breasts should be pumped at regular intervals and the child placed upon some other food until the symptoms are relieved, and then brought back gradually to breast-feeding. Should the infant's digestion be seriously upset a second time as soon as the breast is resumed, the child should be partially or entirely weaned.

If the examination shows the milk to be of very poor quality (i. e., low in fat, low in total solids), whether scanty or abundant, the outlook is not good. It is seldom that the conditions affecting the mother to which such a milk is due can be removed.

When we see a fretful, colicky, sleepless infant with either no gain in weight or a loss of a few ounces a week, and with stools which never approach the normal, and these conditions have lasted for three or four

weeks, we are justified in taking the child from the breast at once (Fig. 27). When the symptoms are less pronounced, and especially when, in spite of all discomfort and indigestion, the infant is gaining in weight,

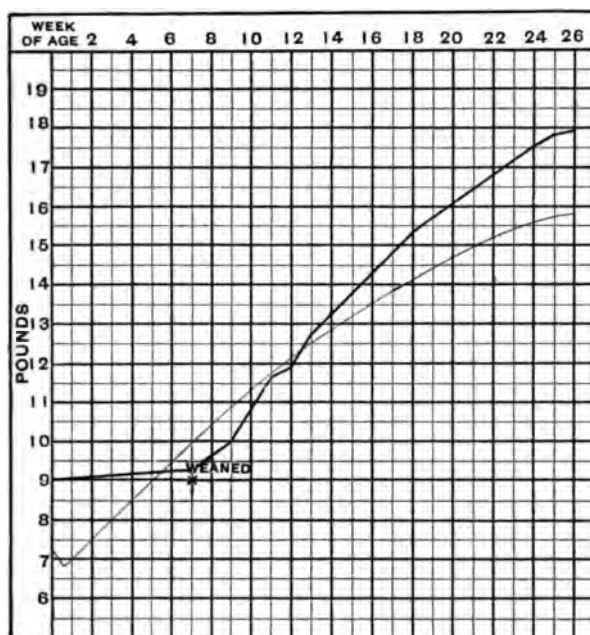


FIG. 27.—WEIGHT CURVE SHOWING THE EFFECT OF BAD NURSING AND GOOD FEEDING. Maternal nursing for seven weeks; continued symptoms of indigestion; colic, frequent green passages, constant discomfort, etc.; other treatment without avail. Immediate improvement when weaned and put on modified milk from the laboratory. Formula: Fat 1.5 per cent, sugar 6 per cent, protein 0.75 per cent. All symptoms of indigestion rapidly disappeared, the percentages were gradually increased, and a steady gain in weight followed.

even though not rapidly, further efforts may be made before weaning is ordered.

*Summary.*—Poor milk is usually low in fat and scanty in quantity, while the protein may be either high or low. Very rich milk is usually high both in fat and protein. Very poor milk can seldom be permanently improved unless the causes are very definite and of a temporary character. Over-rich milk can often be improved if the true explanation for it can be reached. Results are to be judged not so much by the change in the composition of the milk as by improvement in the infant's symptoms. Since good feeding gives so much better results than poor nursing, if circumstances are such that artificial feeding can be properly done, I am inclined to stop nursing after a fair trial—e. g., of two to three weeks—has been made, rather than waste time in prolonged efforts to improve the breast-milk.

**Wet-Nursing.**—In the selection of a wet-nurse, it is by no means so essential as has generally been supposed, that her child shall be of about the same age as the child she is to nurse, for, after the first month, the changes in the composition of breast-milk are insignificant. It is always desirable that the wet-nurse shall have nursed her own infant long enough to demonstrate the fact that she has an abundance of good milk; hence, taking a wet-nurse at the end of the first or second week is always fraught with considerable uncertainty. It is the quality of the milk, not its age, which determines whether or not it will agree. For an infant over one month old, a good wet-nurse whose milk is anywhere between one and six months old will usually answer perfectly well; and even for premature infants such a milk may be used without hesitation, but it should at first be diluted.

A good nurse must, first of all, be a healthy woman, free from syphilitic or tuberculous taint, and her throat, teeth, skin, glands, scalp, and legs should be carefully inspected. She must have good mammary glandular development. The breasts should be full and hard three hours after nursing. They may be very large and yet supply very little milk, being then composed almost entirely of fat. On the other hand, some smaller breasts may be almost all glandular tissue and secrete an abundance of milk. The difference in the size of a breast before and after nursing is one of the best guides as to the amount of milk it is secreting. The nipples should be free from erosions or fissures, and long enough for the needs of the child. Preferably a wet-nurse should be of a phlegmatic temperament, and of a good moral character. This is desirable for personal reasons, although there is no evidence of moral qualities being transmitted through the milk. It is desirable that she should be between twenty and thirty years of age, although much more depends upon the individual than upon the age. Other things being equal, a primipara should be chosen. An examination of the milk may be of some assistance in selecting a nurse; but the best evidence to be obtained of the character of a woman's milk is the condition of her own child, which should always be seen before she is accepted. It often happens that a woman who has had an abundant supply of milk for her own infant has very little for another infant for the first few days in her new surroundings. This is usually the result of the nervous disturbance connected with parting from her own child, going to a new place, being carefully watched, etc. In such a case it should not be too readily decided that she is incompetent as a nurse, for, under most circumstances, with proper treatment the regular flow of milk will be re-established.

**Weaning.**—Weaning should always be done gradually, when possible, for the sake of both mother and child. Sudden weaning is apt to be followed by an attack of acute indigestion in the infant. This, however, is not a necessary result, and usually depends upon the fact that

the child is given cow's milk without sufficient dilution. Weaning in hot weather is usually to be avoided, but the harm from this is not nearly so great as sometimes results where lactation is unduly prolonged because of a prejudice against a change of food at this time. While there are many women of the lower classes who are able to nurse their children to advantage for the entire first year, the number of such among the better classes is certainly very small. By the latter, nursing can rarely be continued beyond the ninth, and often not beyond the sixth month, without unduly draining the vitality of the mother and at the same time harming the child. The late months of lactation, like the early months, require close watching. It is a common mistake to continue both maternal and wet-nursing too long, owing to a dislike of making a change when things are going tolerably. If it has not been done before for reasons previously considered, breast-feeding should be supplemented by other food by the ninth or tenth month in any case. The child's weight is a good guide as to time and amount to be given. In the absence of evident signs of disease, a stationary weight for several weeks makes weaning advisable; a steady loss makes it imperative.

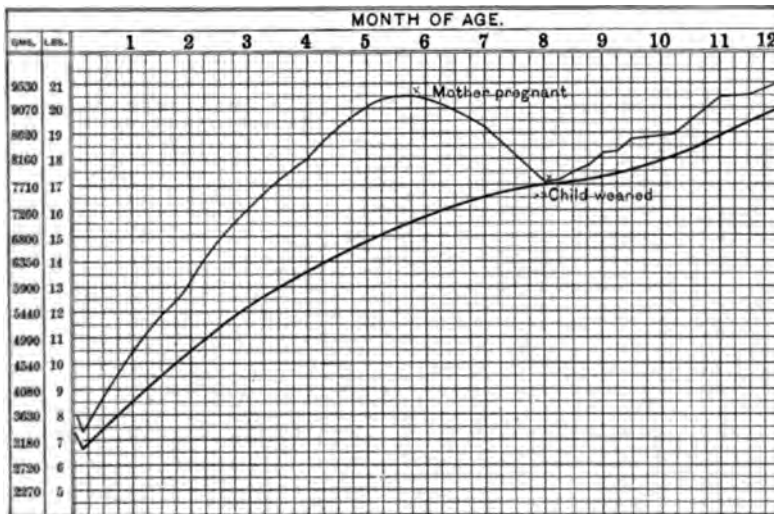


FIG. 28.—CHART SHOWING THE EFFECT OF PREGNANCY UPON THE WEIGHT OF A NURSING INFANT. The upper line is that of the patient; the lower one is the average line for the first year.

The accompanying weight-chart (Fig. 28) illustrates this point. The infant did unusually well until the sixth month. As it did not seem ill, the parents were not disturbed until the loss had reached three pounds. Feeding was at once begun, and the child gradually regained its lost weight. It was subsequently discovered that the mother was pregnant.

When a nursing infant has been accustomed from birth to take one feeding a day from the bottle, always a great convenience to a nursing mother, gradual weaning is generally an easy matter; otherwise it is sometimes an impossibility, the child refusing all food except the breast so long as this is given, and nothing but starvation inducing it to take food either from a bottle or a spoon.

Sudden weaning may be required at any time from the development in the mother of acute disease of a serious nature, such as typhoid fever or pneumonia, of grave chronic disease, such as tuberculosis or nephritis, from the intercurrent of pregnancy, or from disease of the mammary gland. An infant should not be suckled at a breast which is the seat of acute inflammation. Through many of the minor ills—mild attacks of bronchitis, pharyngitis, indigestion, and even malarial fever—mothers frequently nurse their children without any seeming detriment to them or to themselves. In acute illness of short duration, if severe, it is usually better, unless we decide to wean altogether, to feed the child from the bottle and to maintain the flow of milk by the use of the breast-pump three or four times a day rather than to allow it to dry up.

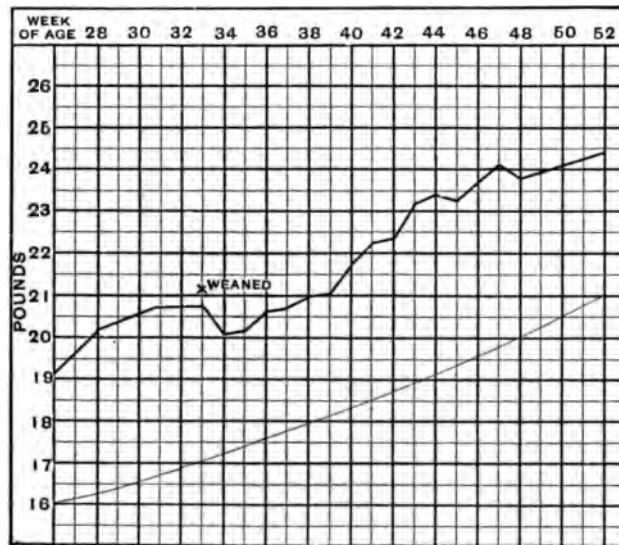


FIG. 29.—WEIGHT CURVE OF A CHILD PROPERLY WEANED. Abrupt weaning at eight months; loss of weight for the first week due to the child's being put upon cow's milk with low percentages. Formula: Fat 1.6 per cent, sugar 6 per cent, protein 0.80 per cent. Percentages were gradually increased, with subsequent steady and regular gain in weight. Weaning accomplished without the slightest symptom of indigestion. The lower is the average line.

In cases of sudden weaning, the food should in the beginning be very much weaker than for an artificially fed child of the same age. The change can then be made without causing much disturbance (Fig. 29).

When the infant has become somewhat accustomed to cow's milk the strength of the food may be gradually increased.

The difficulties in weaning a child who up to nine or ten months has had no food but the breast are sometimes great. Much time and tact are necessary on the part of both physician and nurse in these cases. To try to teach older infants to take the bottle is unwise; feeding from cup or spoon is usually quite as easy. Continued coaxing of food is objectionable; forcing is much worse and prolongs the struggle. In my experience I have found the best way to offer food at regular intervals and to take it away at once if refused. This is repeated every three or four hours. A variety of things may be offered—modified cow's milk, thick gruels, beef juice, broths, bread and milk, etc. The nature of the food seems to make very little difference. A strong-willed child will often hold out for twenty-four or thirty-six hours, and occasionally a very stubborn one is found who will do so for forty-eight hours. At the end of this time the pangs of hunger are generally so acute that he capitulates. Serious symptoms from withholding food under such circumstances I have never seen.

#### MIXED FEEDING.

By mixed feeding is meant a combination of nursing and artificial feeding. There are no objections to this practice; on the contrary, there are great advantages in giving an infant only a few breast-feedings a day when more are impossible. This may frequently be done in hospital practice, and thus a single wet-nurse may assist in the feeding of several infants. Mixed feeding may be resorted to whenever the milk supply of the mother is insufficient. If at any time the mother's health begins to suffer, she may be relieved of night nursing or of one or more nursings during the day, and the bottle substituted. In this way she may be enabled to continue lactation for some time longer than would otherwise be possible. Mixed feeding is often necessary during the first few weeks, while the mother's milk is insufficient in consequence of something which has retarded her convalescence. For the advantage of the stimulation to secretion afforded by the child's nursing, it is usually better, rather than alternate the breast and the bottle, to put the child at first to the breasts. After he has emptied them, additional food may be given from the bottle if the baby is still hungry. The milk may become abundant and of good quality as soon as the mother is well enough to be up and out of doors, although it was previously scanty and of inferior quality. Two or three feedings a day from the bottle help to bridge over this period and prevent the child's nutrition from suffering. But before allowing a mother partly to nurse and partly to feed her infant, one should be sure that the quality of her milk is good.



It is well from the very outset to accustom the infant to take one feeding from a bottle each day. In maternal nursing, the occasional feeding which is usually necessary becomes then a simple matter. If the child is being wet-nursed, the same plan is advisable, for it is then easy to put an infant upon the bottle entirely in the event of the wet-nurse leaving suddenly—a not uncommon occurrence.

#### ARTIFICIAL FEEDING.

There are a few fundamental principles regarding which nearly all pædiatrists are agreed.

Woman's milk is not only the best, it is the ideal infant food.

Any substitute should furnish the same constituents—fat, carbohydrates, protein, salts, and water, and in sufficient quantities to supply the needs of the body for its nutrition and growth;<sup>1</sup> furthermore, they should be in about the same proportion as they exist in a good sample of woman's milk.

The different constituents should resemble those of woman's milk as nearly as possible both in their chemical composition and in their behaviour toward the digestive fluids.

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<sup>1</sup> From numerous observations, the nutritive needs of the average infant in health have been shown to be about 100 calories for each kilo. of body weight from the third week to the sixth month. These gradually diminish until at the end of the first year they reach about 75 to 80 calories per kilo. The caloric requirements are greater for very active infants on account of their more rapid metabolism; also, for premature infants or those much below average weight, on account of their relatively larger body surface to radiate heat. For such infants from 125 to 150 calories per kilo. may be necessary.

An infant weighing 7 kilos. (15 pounds) requires about 700 calories daily. As the caloric value of a good average specimen of woman's milk is about 650 calories per litre, the requirements would be supplied by a little over one litre of woman's milk.

The practical application of these facts in infant-feeding is that one should be careful to furnish to an infant who is artificially fed what is needed, but no considerable excess. A food much below the normal caloric requirements, or one much above them, may be equally improper and therefore unsuccessful. The physician should be able to calculate the caloric value of the food given, to see if possible where the mistake lies, when infants are not thriving.

The caloric value of any modification of cow's milk of known percentages may be calculated as follows: An infant is taking six feedings of 6 ounces, or 36 ounces daily of a milk containing, fat 3.5 per cent, sugar 7 per cent, protein 1.75 per cent.

$$\begin{aligned} .035 \text{ (fat \%)} \times 9.3 \text{ (cal. val. o' fat)} &= .325 \text{ cal. val. of fat in 1 grm. food.} \\ .07 \text{ (sugar \%)} \times 4.1 \text{ (" " " sugar)} &= .287 \text{ " " " sugar " 1 " " } \\ .0175 \text{ (protein \%)} \times 4.1 \text{ (" " " protein)} &= .072 \text{ " " " protein " 1 " " } \\ &= .684 \text{ caloric value of 1 gram of food.} \end{aligned}$$

$$.684 \times 1000 = 684 \text{ (caloric value 1 litre food).}$$

$$36 \text{ ounces} = 1.06 \text{ litres: } 1.06 \times 684 = 725 \text{ (No. of calories in food taken daily).}$$

Such calculations may be applied to any milk formula of known percentage, but are rather laborious. A simpler way of arriving at the same result is to multiply the

No food except fresh milk from some other animal meets the requirements even approximately.

In the artificial feeding of infants, cow's milk is selected as being the only milk available for general use. Although it furnishes all the constituents required, they are not present in the proportions suited to young infants, and the constituents are not identical with those in woman's milk. Cow's milk, therefore, can not be fed to most infants without some changes. These changes are technically known as the *modification of cow's milk*. To make these changes properly it is necessary to know what are the difficulties in the digestion of cow's milk and how these may be overcome.

The earliest milk modification was simply dilution with water and the addition of enough cane sugar to make it taste like breast-milk. The only change made with the age of the child was simply to vary the amount of water. Instead of water as a diluent many preferred to use gruels made from different cereals, believing that thereby the casein was rendered more digestible. Upon such simple modifications as these many children have done, and many still do, very well, when the matter of dilution is judiciously managed. But it is equally true that present knowledge enables us to do something better. There are, however, circumstances in which anything more complex is impossible in the way of milk modification.

Later, when the composition of woman's milk came to be better understood, it was thought that all that was necessary in modified milk was to secure the exact percentages of fat, protein, sugar, and salts which exist in a good sample of woman's milk, and that this combination would be the best possible substitute for it. Out of this came the

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caloric values of the different ingredients used in making up the food by the amount of each one that is taken. These values are approximately as follows:

|   |                        |       |
|---|------------------------|-------|
| 1 ounce 7 per cent milk                 | has a caloric value of | 27.5  |
| 1 " 6 " " "                             | " " " " "              | 25.0  |
| 1 " 5 " " "                             | " " " " "              | 22.5  |
| 1 " 4 " " "                             | " " " " "              | 20.0  |
| 1 " 3 " " "                             | " " " " "              | 17.5  |
| 1 " 2 " " "                             | " " " " "              | 15.0  |
| 1 " 1 " " "                             | " " " " "              | 12.5  |
| 1 " fat-free "                          | " " " " "              | 10.0  |
| 1 " whey "                              | " " " " "              | 10.0  |
| 1 " milk sugar by weight                | " " " " "              | 116.0 |
| 1 " " " " volume                        | " " " " "              | 72.0  |
| 1 even tablespoonful of milk sugar      | " " " " "              | 44.0  |
| 1 ounce barley flour by weight          | " " " " "              | 100.0 |
| 1 " " water (1 tablespoonful to a pint) | " " " " "              | 2.0   |
| 1 " malt-soup extract                   | " " " " "              | 80.0  |
| 1 " condensed milk                      | " " " " "              | 132.0 |
| 1 " olive oil by volume                 | " " " " "              | 245.0 |

various mixtures of milk, cream, sugar, etc., which aimed to reproduce, according to the views of different writers, the exact proportions of woman's milk. This was a step in advance, in that some proper relation between the different food constituents was maintained. Experience, however, has shown that no single milk-formula can serve as a substitute for woman's milk; and intelligent students of the problem have ceased to search for one.

In the percentage method of infant-feeding one considers the different elements of the food separately and tries to adapt their proportions to the child's digestion. While it is based upon the percentage composition of woman's milk, it recognises that there are differences in the digestibility of cow's milk and woman's milk. It aims to discover the proper proportions of fat, sugar, and protein, and the best methods of gradational increase for healthy infants with normal digestion; and also to discover for those with abnormal or feeble digestion, the combinations best suited to the individual conditions. Percentages are simply a method of stating definitely the composition of the food which we are giving. There is, therefore, strictly speaking, no such thing as the percentage method of feeding; it is merely a method of statement.

For the fundamental work along this line we are indebted to Prof. T. M. Rotch, of Harvard, and Mr. C. E. Gordon, of the Walker-Gordon Laboratory Company.

The calculation of food requirements of an infant in terms of calories is at present much employed. The requirements are assumed to be fairly uniform for an infant of a given weight (the figures are given on a previous page), and for healthy, well-nourished children this is approximately correct. But the calculation is not correct for those who are below the average weight for their ages. For such children the food requirements measured in calories are considerably greater than those allowed by the theoretical calculation. A comparison of the physiological requirements as calculated, with the calories furnished by the food given, is a useful method of control in the feeding of children who are not thriving or whose nutrition is especially difficult. It enables one to see whether he is feeding far above or far below physiological requirements, and also to appreciate the necessity of increasing some elements in the food if others are reduced. It may be regarded as a basis of calculating food requirements, but nothing more. Like the percentage method, it is a method of statement; the two are not in contrast or opposition, and both are valuable.

**The Modification of Cow's Milk for Healthy Infants during the First Year.**—It is absolutely necessary to consider separately the changes required by healthy infants with normal digestion, those required by infants with feeble digestion, and those required by infants suffering from more or less indigestion. From a failure to make this distinction, much

confusion has arisen. The digestion of all healthy infants is very much alike, and they can all be fed in much the same way; while, on the contrary, the variations afforded by unhealthy infants are almost endless, and each case must be considered by itself. If it is only healthy infants that can be fed by rule, it is equally true that if fed from the beginning by proper rules most infants will remain healthy.

In adapting cow's milk for infant-feeding we must realise at the outset that, no matter how we may alter it, cow's milk is not a perfect substitute for woman's milk. It should not be lost sight of that there are inherent differences which will never be altogether removed. The following table gives the proportions of the various elements which make up the two milks:

|               | Woman's milk,<br>average. | Cow's milk,<br>average. |
|---------------|---------------------------|-------------------------|
|               | Per cent.                 | Per cent.               |
| Fat .....     | 3.50                      | 4.00                    |
| Sugar .....   | 7.00                      | 4.50                    |
| Protein ..... | 1.50                      | 3.50                    |
| Salts .....   | 0.20                      | 0.75                    |
| Water .....   | 87.80                     | 87.25                   |
|               | 100.00                    | 100.00                  |

These quantitative differences in the constituents are important. It will be seen that cow's milk has an excess of protein and salts, but is deficient in sugar. Far more important, however, for the infant are the qualitative differences. The sugar in the two milks, it is true, is nearly if not quite the same. The fat of cow's milk, however, contains a smaller proportion of oleic acid and a much larger proportion of volatile fatty acids. The salts are excessive in amount, particularly calcium phosphate, but are deficient in iron and potassium. There are important differences in the protein. The total protein of cow's milk is nearly two and a half times as great as that in woman's milk. In cow's milk the soluble protein (lactalbumin, etc.) is only about one-third or one-fourth as abundant as the insoluble protein (casein); while in woman's milk the soluble protein forms more than half the total. But the difference in the digestibility of the protein of the two milks is much less than was once believed. Other important conditions relate to the reaction of milk, its freshness, bacterial contamination, etc. The modification of milk must aim, therefore, at something more than overcoming the quantitative differences in the constituents.

In the adaptation of cow's milk for infant-feeding the emphasis has been at different times laid upon different elements. The view was long held that the chief trouble was with the protein. As a result of this the use of predigested milk came largely into vogue, and milk formulas

with high fat and low protein were widely employed. Then came the opinion prominently advanced by Czerny and Keller that it was the fat which produced the most trouble. More recently it has been pointed out by Finkelstein and his pupils that disturbances of the gravest character may be due to the sugar and even to the salts. Our knowledge on this subject leaves many points still unsettled. Meanwhile, the important thing for the student and the practitioner to appreciate, is the fact that any of the elements of cow's milk may cause serious disturbance. For the healthy child we are safe in emphasising that trouble is most likely to be due to the fat, while one with disordered digestion may be disturbed by any one of the elements. However, one must be careful about inferring, from the disturbances in sick infants, how healthy ones are to be fed.

**FAT.**—The amount of fat of cow's milk which a healthy infant can digest varies considerably; the usual limits are between 1 and 4 per cent. There are not many infants who can digest as much fat of cow's milk as the proportion often present in a good sample of breast-milk. With most infants it is necessary to begin with as low a proportion as 1 per cent. The increase should be made very gradually. I have not found it advantageous to increase the fat above 4 per cent; for most infants under usual conditions the upper limit should not be over 3.5 per cent. I constantly see serious derangements of digestion produced by the use of higher percentages.<sup>1</sup>

The danger of disturbing the infant's digestion by fat has only recently been sufficiently appreciated. This mistake is frequently made when rich Jersey milk is employed, and also when the fat percentage is steadily raised for the purpose of overcoming chronic constipation. For nearly all infants with disordered digestion the fats must be much reduced. No modification of the fat of cow's milk is possible except in the amount. There seems to be no difference in the digestibility of gravity and centrifugal cream. Freshness is a very important consideration in all extra fat added to milk.

**SUGAR.**—In woman's milk the percentage of sugar varies but little; it is usually between six and seven per cent. In feeding cow's milk it is seldom necessary to have the sugar less than five or more than seven per cent. To obtain the proper proportion of sugar is the simplest part of the modification. It is only necessary to calculate the amount to be added to bring this up to the per cent desired. While, for reasons given elsewhere, lactose is the form of sugar generally preferred, when this can not be obtained, cane sugar may be substituted, but in a somewhat smaller amount. Besides, there is some difference in the digestibility of these two sugars. In certain forms of intestinal indigestion

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<sup>1</sup> Archives of Pædiatrics, January, 1905.

cane sugar is sometimes better tolerated than is milk sugar. Maltose also may be used; it possesses certain advantages as well as disadvantages, which should be carefully considered before it is employed. It should be distinctly understood that the purpose of adding sugar is not to sweeten the food, but to furnish the proper proportion of soluble carbohydrates for nutrition.

**PROTEIN.**—To the modification of the protein of cow's milk most of the attention was formerly given. The evidence seems conclusive, however, that healthy infants digest this protein without difficulty. The main point necessary therefore is to decide upon the quantity which shall be given.

During the early weeks not more than one per cent of protein is required. The amount should be gradually increased so that an average child will receive at four or five months two per cent of protein and three per cent at eight or nine months. It is a common mistake to continue long with too low protein. Anæmia, malnutrition, and, I believe, sometimes scurvy are seen as a consequence of this practice. The gradual increase is therefore just as important as the low beginning.

**INORGANIC SALTS.**—These may generally be calculated in cow's milk as one-fifth the total protein. When the total protein has been suitably reduced by dilution the amount of total salts will approximate that present in woman's milk. But it should not be forgotten that such dilution, while it brings down those salts which are in excess, chiefly calcium phosphate, to a proper proportion, also reduces to the same degree the iron and potassium which originally were not in excess. The influence of the inorganic salts upon nutrition is something deserving further study. In certain pathological conditions the salts are undoubtedly capable of producing serious disturbances.

The amount of reduction obtained by the different dilutions is shown in the following table:

|                       | Cow's milk. | Diluted once. | Diluted twice. | Diluted 3 times. | Diluted 4 times. |
|-----------------------|-------------|---------------|----------------|------------------|------------------|
| Protein .....         | 3.50        | 1.75          | 1.16           | 0.87             | 0.70             |
| Inorganic salts ..... | 0.75        | 0.37          | 0.25           | 0.18             | 0.15             |

**REACTION.**—It has been customary to overcome the excessive acidity of cow's milk by adding either lime-water or bicarbonate of soda. Of the former, there is generally employed about one ounce to each twenty ounces of the food; of the latter, about one grain to each ounce of the food. The manner in which the addition of these substances affects the digestion of milk is not fully understood. The practical value of adding lime-water is well established by clinical experience. Some recent experiments of T. W. Clarke indicate that its chief effect may be due to

its stimulation of the secretion of hydrochloric acid. Lime-water also causes a retardation of coagulum formation in the stomach.

**BACTERIA.**—These are always present in cow's milk. They have been already considered in the pages devoted to the Sterilisation of Milk.

**THE OBSERVATION OF CASES OF INFANT-FEEDING.**—For the first few weeks it is essential that the physician see the infant every few days, inspect the stools, hear the nurse's report, and see how his directions are being carried out. When the child is well started and has begun to gain regularly in weight, a weekly visit will be sufficient. Still later a regular weekly report in writing, to be continued up to the seventh or eighth month, may be all that is required; after that time monthly reports are usually sufficient. My plan is to have the weekly report include only answers to certain questions, viz.:

1. Weight:.....gain or loss since last report.
2. Stools: frequency and general character.
3. Vomiting or regurgitation—when? and how much?
4. Flatulence or colic?
5. Appetite: Is the child satisfied? Does he leave any of his food?
6. Is he comfortable and good-natured and sleeping well?
7. The formula of the food now given; quantity and frequency of feedings.
8. Date.
9. Date of last report.

An excellent plan is to furnish the patient with printed forms containing these questions to be filled out and returned. This is a simple matter, and there are very few intelligent mothers who will be unwilling to co-operate with the physician to this extent. With information regarding the points indicated, it is possible for the physician to know pretty accurately how the case is doing, what changes, if any, are desirable in the food, and whether he ought to see the patient. It is only by some systematic method of observation that one can secure the best results with any form of infant-feeding.

**MILK LABORATORIES.**—The first milk laboratory was established in Boston by the Walker-Gordon Company in 1892; one in New York in 1893, and since that time others in many American cities. They undertake to furnish "modified milk" of any desired proportions, upon the prescription of physicians. The elements chiefly used by the Walker-Gordon laboratories are: (1) Cream containing 32 per cent of fat; (2) separated milk, from which the fat has been removed by the centrifugal machine; (3) a standard solution of milk sugar, 20 per cent strength. These contain fat, sugar, and protein in the following proportions:

|              | Cream.    | Separated milk. | Sugar solution. |
|--------------|-----------|-----------------|-----------------|
|              | Per cent. | Per cent.       | Per cent.       |
| Fat.....     | 32.00     | 0.05            | .....           |
| Sugar.....   | 3.40      | 5.00            | 20.00           |
| Protein..... | 2.50      | 3.55            | .....           |

By combining these it is possible to vary the percentages of fat, sugar, and protein in the milk to almost any degree desired, and to do this with very great accuracy. By using whey, a separate modification of the protein is accomplished; so that within certain limits a larger proportion of whey protein, chiefly lactalbumin, can be given. The highest proportion of whey protein with the lowest proportion of casein can be given when the total protein does not exceed 1.15 per cent; of this, 0.90 per cent may be whey protein and 0.25 per cent casein. The alkalinity is usually obtained by adding lime-water in any desired amount. The laboratory adds, when requested, gruels of wheat, oats, or barley of any desired strength; and, finally, it delivers the milk raw, or heats it for sterilisation to any temperature ordered by the physician.

The food-supply for the entire day is delivered each morning in the bottles from which it is to be fed. The empty bottles returned are washed and sterilised at the laboratory. In ordering the food the physician simply writes for the percentages of fat, sugar, and protein which he desires, together with the number of feedings for twenty-four hours and the quantity for each feeding:

|                              |             |
|------------------------------|-------------|
| Fat.....                     | 2 per cent. |
| Sugar.....                   | 6 "         |
| Protein.....                 | 1 "         |
| Alkalinity, lime-water.....  | 5 "         |
| Number of feedings.....      | 8           |
| Amount for each feeding..... | 4 ounces.   |
| Heat to 155° F., 30 minutes. |             |

The aim of the laboratory is to supply the physician with any milk modification which he may desire to use and to do this with accuracy.

One is not restricted to any method or plan of feeding, but can carry out his own method with much greater accuracy than is possible when the milk is prepared in the average home. He is independent of the ignorance, carelessness, or caprice of the nurse who otherwise would probably prepare the food. But by whatever method the child is fed the physician who assumes the responsibility to direct must be familiar with the subject and he must keep in touch with the case if he expects good results.

As a general guide to the modification of milk for an average healthy infant the following table is introduced, showing the manner in which the changes required by the development of the child may be made:



*Table showing percentages of fat, sugar and protein which may be ordered from the Milk Laboratory and are suitable for healthy infants for the first year.*

|                         | Fat. | Sugar. | Protein. | Whey protein. | Casein. |
|-------------------------|------|--------|----------|---------------|---------|
| <b>Weak Formulas.</b>   |      |        |          | or            | and     |
| I.                      | 0.75 | 4.00   | 0.75     | 0.70          | 0.05    |
| II.                     | 1.00 | 5.00   | 0.75     | 0.70          | 0.05    |
| III.                    | 1.00 | 5.00   | 1.00     | 0.85          | 0.15    |
| IV.                     | 1.25 | 5.00   | 1.00     | 0.85          | 0.15    |
| V.                      | 1.50 | 5.00   | 1.25     | 0.80          | 0.45    |
| <b>Medium Formulas.</b> |      |        |          |               |         |
| VI.                     | 1.75 | 6.00   | 1.50     |               |         |
| VII.                    | 2.00 | 6.00   | 1.50     |               |         |
| VIII.                   | 2.25 | 6.00   | 1.75     |               |         |
| IX.                     | 2.50 | 6.00   | 1.75     |               |         |
| X.                      | 2.75 | 6.00   | 1.75     |               |         |
| XI.                     | 3.00 | 6.00   | 2.00     |               |         |
| <b>Strong Formulas.</b> |      |        |          |               |         |
| XII.                    | 3.25 | 6.00   | 2.00     |               |         |
| XIII.                   | 3.50 | 6.00   | 2.25     |               |         |
| XIV.                    | 3.50 | 6.00   | 2.50     |               |         |
| XV.                     | 3.50 | 6.00   | 3.00     |               |         |
| XVI.                    | 4.00 | 4.50   | 3.50     |               |         |

(Whole Milk.)

The first group, classed as weak formulas, are designed for normal infants during the first few weeks, or for those with feeble digestion, of whatever age.

The second group is designed for the needs of normal infants from about one month to four or five months, although there are many who can not take a stronger food for a much longer time.

The third group is expected to cover, for children with good digestion, the period from about the fifth month to the twelfth or thirteenth month, gradually leading up to whole milk.

It is important to begin with a weak formula for a young infant, and for one with feeble digestion, whatever its age. One may then gradually increase the strength of the milk according to the indications afforded by the child's appetite and powers of digestion. With some the increase can be made more rapidly than with others, but with all children it is important that the steps of increase should be gradual and not greater than are indicated in the formulas of the table; it may even be desirable at times to make them more slowly than is there suggested. In the table the total protein to be used is indicated and also the quantities of whey protein and casein, when one desires to order these separately. There is some advantage in so dividing the protein for very young or premature infants.

**Home Modification of Milk.**—For the great majority of infants the milk is necessarily prepared at home. No plan of home modification yet

proposed secures more than approximate accuracy in the percentages of fat, sugar, protein, etc.; yet, if the directions given below are carefully carried out, a degree of accuracy sufficient for all practical purposes can be secured. The physician thus can not only know the percentages he is giving, but he can himself readily vary them within the range usually required, according to the indications presented. The thing desired is a method simple enough to be readily grasped by the average mother or nurse who is to carry out the physician's directions.

The requisites for success in the home modification of milk are:

Good raw materials—the freshest and cleanest milk obtainable.

Knowledge on the part of the physician of at least the fat content of the milk and cream used in the home as it is only the fat which is subject to much variation.

Directions which are clear, explicit, and in writing, that they may not be misunderstood.

The co-operation of an intelligent mother or nurse, that they may be properly carried out.

**HOW TO OBTAIN FORMULAS REQUIRED FOR GENERAL USE.**—A considerable variety of formulas is required. For normal children with good digestion the fat should usually be higher than the protein, the upper limit being twice as much fat as protein. For those with disturbances of digestion, the fat should usually be lower than the protein. A series of formulas, with the range required, can readily be obtained by the method given below. Nearly all who practise home modification of milk purchase milk in quart bottles.<sup>1</sup> This has therefore been made the basis of calculation. If the milk used has 4 per cent of fat and the directions given are closely followed the results obtained will be very nearly accurate.

The first step is to secure milks containing definite amounts of fat varying from 7 per cent down to 1 per cent. This has been described in detail for 4- and 5-per-cent milk in the chapter on Cow's Milk.

It is convenient to calculate all food formulas on a basis of a 20-ounce mixture.

Every ounce of 7% milk in 20-oz. mixture has one-twentieth of 7, or 0.35% fat.

|   |   |   |    |   |   |   |   |   |   |    |   |       |   |
|---|---|---|----|---|---|---|---|---|---|----|---|-------|---|
| " | " | " | 6% | " | " | " | " | " | " | 6, | " | 0.30% | " |
| " | " | " | 5% | " | " | " | " | " | " | 5, | " | 0.25% | " |
| " | " | " | 1% | " | " | " | " | " | " | 1, | " | 0.05% | " |

The percentage of protein and sugar in the various milks we are considering differs so little that the variation may be ignored. Since

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<sup>1</sup> If instead of purchasing milk in bottles milk fresh from the cow is used, as soon as received it should be strained through three thicknesses of cheesecloth or a layer of absorbent cotton into quart jars or milk bottles, and allowed to stand in ice-water or cold spring water for at least four hours. The top milk may then be removed.

*Formulas obtained from milk containing different percentages of fat.*

|      |                       | A<br>7%<br>Milk. | B<br>6% | C<br>5% | D<br>4% | E<br>3% | F<br>2% | G<br>1% | with Protein | Per<br>cent. | Per<br>cent. |
|------|-----------------------|------------------|---------|---------|---------|---------|---------|---------|--------------|--------------|--------------|
| I    | 1 ounce in 20 has Fat | 0.35             | 0.30    | 0.25    | 0.20    | 0.15    | 0.10    | 0.05    | 0.175        | 0.225        |              |
| II   | 2 ounces "            | 0.70             | 0.60    | 0.50    | 0.40    | 0.30    | 0.20    | 0.10    | "            | 0.45         | Sugar        |
| III  | 3 " "                 | 1.05             | 0.90    | 0.75    | 0.60    | 0.45    | 0.30    | 0.15    | "            | 0.65         | "            |
| IV   | 4 " "                 | 1.40             | 1.20    | 1.00    | 0.80    | 0.60    | 0.40    | 0.20    | "            | 0.90         | "            |
| V    | 5 " "                 | 1.75             | 1.50    | 1.25    | 1.00    | 0.75    | 0.50    | 0.25    | "            | 1.10         | "            |
| VI   | 6 " "                 | 2.10             | 1.80    | 1.50    | 1.20    | 0.90    | 0.60    | 0.30    | "            | 1.35         | "            |
| VII  | 7 " "                 | 2.45             | 2.10    | 1.75    | 1.40    | 1.05    | 0.70    | 0.35    | "            | 1.55         | "            |
| VIII | 8 " "                 | 2.80             | 2.40    | 2.00    | 1.60    | 1.20    | 0.80    | 0.40    | "            | 1.80         | "            |
| IX   | 9 " "                 | 3.05             | 2.70    | 2.25    | 1.80    | 1.35    | 0.90    | 0.45    | "            | 2.00         | "            |
| X    | 10 " "                | 3.50             | 3.00    | 2.50    | 2.00    | 1.50    | 1.00    | 0.50    | "            | 2.25         | "            |
| XI   | 11 " "                | 3.80             | 3.30    | 2.75    | 2.20    | 1.65    | 1.10    | 0.55    | "            | 2.45         | "            |
| XII  | 12 " "                | —                | 3.60    | 3.00    | 2.40    | 1.80    | 1.20    | 0.60    | "            | 2.70         | "            |
| XIII | 13 " "                | —                | 3.90    | 3.25    | 2.60    | 1.95    | 1.30    | 0.65    | "            | 2.90         | "            |
| XIV  | 14 " "                | —                | —       | 3.50    | 2.80    | 2.10    | 1.40    | 0.70    | "            | 3.15         | "            |
| XV   | 15 " "                | —                | —       | —       | 3.00    | 2.25    | 1.50    | 0.75    | "            | 3.35         | "            |

From 4% Milk. .... From 5% Milk.

To obtain 7% milk use upper 16 ounces ..... upper 20 oz. from one quart.

" " 6% " " 20 " " " 24 oz. " "

" " 5% " " 24 " " " all

" " 4% " " all ..... remainder after skimming off 2 oz.

" " 3% " " remainder after skimming off 2 oz. " " 3

" " 2% " " " " 4 " " " 5

" " 1% " " " " 8 " " 8

With Formulas I to V, enough sugar should be added to raise the amount to 5 per cent.

With Formulas VI to XV, enough sugar should be added to raise the amount to 6 per cent.

4-per-cent milk contains 4.50 per cent of sugar, each ounce of any of these milks in a 20-ounce mixture will have one-twentieth of 4.50 or 0.225 per cent of sugar. Each ounce in a 20-ounce mixture will have one-twentieth of 3.50, or 0.175 per cent of protein. The figures given in the accompanying table will now be clear. The table shows the percentage composition of the different formulas containing twenty ounces, which can be derived from the different milks and the manner in which they are obtained. It should be emphasised that in general, formulas from 7-, 6-, 5-, and 4-per-cent milk are to be used for healthy infants with good digestion; formulas from 3-, 2-, and 1-per-cent milk are to be used for infants suffering from disorders of digestion.

These formulas cover practically all our needs. This table may seem at first glance somewhat complicated; but it is not so if we observe that column A, for instance, gives the fat percentage of the food when one, two, three or more ounces of a 7-per-cent milk are used in a 20-ounce mixture; column B gives the same when a 6-per-cent milk is used, etc.

From left to right the table would, therefore, read as follows: Taking Formula VIII, eight ounces in twenty has 2.80 per cent of fat if 7-per-cent milk is used; 2.40 per cent of fat, if 6-per-cent milk is used, etc., with, in every case, 1.40 per cent of protein and 1.80 per cent of sugar. It will be noted that the protein and sugar percentages remain the same whichever percentage of fat, from 7 per cent to 1 per cent, the milk contains from which the formula is made up. It is thus evident how one may vary the fat without varying the protein and sugar.

Thus far only the protein and fat have been considered. To secure the desired percentage of sugar is a simple matter. One notes first the percentage of sugar contained in the milk after dilution; subtracting this from the percentage desired will give the percentage to be added.<sup>1</sup>

Thus, if we use ten ounces in twenty of milk containing any of the percentages of fat from 7 to 1, the sugar present in the mixture is 2.25. To raise this to 6 per cent, one must add 3.75 per cent, or a little over two even tablespoonfuls, to each twenty ounces of the mixture. The sugar should be dissolved in the diluent before adding to the milk.

The usual proportion of lime-water added is 5 per cent, or one ounce in a 20-ounce mixture; this may be increased to any desired quantity.

The quantity of the diluent must in each instance be sufficient to bring the total up to twenty ounces. As a diluent for the early months plain boiled water is generally to be preferred. After five or six months barley or oatmeal water may be substituted.

To make more than a 20-ounce mixture will be found simple if one

<sup>1</sup> One ounce of milk sugar by weight in a 20-ounce mixture adds 5 per cent.

" " " " " " volume " " " " " about 3 per cent.

" even tablespoonful " " " " " " 1.75 per cent.

calculates for 25, 30, 35 ounces, etc. Thus, for 25 ounces there is added one-fourth more of each ingredient; for 30 ounces, one-half more, etc.

**THE APPLICATION OF THE FOREGOING FORMULAS IN PRACTICE.—GENERAL RULES FOR VARYING MILK PERCENTAGES.**—We have indicated in the paragraph upon laboratory feeding a series of formulas suitable for the first year, and have shown how similar formulas can be obtained when the milk is prepared at home. A schedule like that given in the table is useful to indicate in a general way what percentages an average infant may be expected to take. But no schedule can be closely followed with any given child. One can not conclude that because a child is six weeks old he is able to digest milk containing certain percentages, and certain others because he is six months old. To attempt to follow a schedule too closely is to violate the fundamental principle of percentage feeding, which is to adapt the milk to the child's requirements and powers of digestion at any time. In brief, one should begin with weak formulas and gradually increase their strength according to the child's needs and his ability to digest cow's milk.

Although it is impossible to follow a schedule in regard to the composition of the food for the first year, one may generally with advantage follow a schedule with regard to quantity and frequency of feeding.

*Schedule for Healthy Infants during the First Year.*

| AGE.                          | Interval between meals, by day. | Night feedings 10 P.M. to 7 A.M. | No. of feedings, 24 hours. | Quantity for one feeding. |          | Quantity for 24 hours. |             |
|-------------------------------|---------------------------------|----------------------------------|----------------------------|---------------------------|----------|------------------------|-------------|
|                               |                                 |                                  |                            | Ounces.                   | Grammes. | Ounces.                | Grammes.    |
| 2d to 7th day . . . . .       | 2                               | 2                                | 10                         | 1-1½                      | 30-45    | 10-15                  | 300-450     |
| 2d to 4th week . . . . .      | 2½                              | 1                                | 8                          | 1½-3½                     | 45-110   | 12-28                  | 360-875     |
| 5 weeks to 2 months . . . . . | 3                               | 1                                | 7                          | 3-5                       | 90-155   | 21-35                  | 630-1,085   |
| 2 to 5 months . . . . .       | 3                               | 1                                | 7                          | 4-6                       | 125-185  | 28-42                  | 875-1,300   |
| 5 to 9 months . . . . .       | 3                               | 0                                | 6                          | 5-7½                      | 150-235  | 30-45                  | 900-1,400   |
| 9 to 12 months . . . . .      | 4                               | 0                                | 5                          | 7-9                       | 220-280  | 35-45                  | 1,085-1,400 |

*How and Where to Begin.*—With young infants having presumably normal digestion it is desirable to begin with weak formulas, such as No. V of C or D, with sugar raised to 5 or 6 per cent.

The same strength should be used for a few days to test the child's digestion. For a healthy infant of eight pounds weight, two weeks old, one should begin with 2½ ounces at a feeding and feed eight times a day, interval between feedings two and a half hours. The quantity for one feeding can soon be increased to 3, then to 3½ ounces.

For a smaller or less vigorous child, one should begin with No. IV of C or D and give 1½ or 2 ounces at the same intervals, increasing the quantity, however, more slowly.

For a healthy child with normal digestion, weaned at four or five

months, one should begin with No. VI of B or C and give a larger quantity, i. e.,  $4\frac{1}{2}$  to 6 ounces at three-hour intervals, and increase the strength more rapidly than with a younger infant.

For one weaned at nine or ten months one should begin with No. VII of B or C, 6 or 7 ounces at a feeding and increase both strength and quantity rather rapidly.

A stationary weight for a week or two, or even a loss of a few ounces, is of no importance, provided the change in diet can be effected without deranging digestion; for as soon as a child becomes accustomed to cow's milk the percentages can be raised, and progress is assured. Nothing is easier than to derange the digestion in the beginning by the use of too strong food; such disturbances, though they may not be severe, often continue for many weeks (Fig. 30). The closest attention is required in the beginning. If a good start is made, subsequent progress is easy; but with a bad start there is likely to be trouble most of the time. As soon as an infant's capacity to digest cow's milk is ascertained, the food can be increased accordingly.

*Indications for Increasing the Food.*—While it is important to begin with low percentages, it is a serious mistake to continue long with them. The power of digestion is strengthened by gradually increasing the work the organs are given to do. Abrupt increases are almost certain to disturb digestion. A proper rate of increase of the fat and protein is indicated in the table of formulas reading downward in the different columns.

How rapidly the increase is made will vary much with the individual infant. With a vigorous child, above average weight, with good digestion, the strength may be increased rather rapidly, and also the quantity given at one feeding. With a small or delicate child, or one with feeble digestion, one must advance much more slowly both with respect to the strength and quantity of food. No greater mistake can be made than to attempt to measure the increase in food by the age of the child. We can not raise the percentages every week or every month regardless of other conditions. The progress in weight is important, yet one should not be guided by it alone in increasing the food. With the weak food necessary at first no material gain in weight is to be expected. However, if there is no vomiting or colic, if the child is entirely comfortable and sleeps most of the time, and if the stools have a normal colour and odour, conditions may be considered satisfactory. The food may be cautiously strengthened with the demands of the child's appetite, and soon the increase in weight will begin, and when once begun it is likely to continue. On the contrary, if the weight is made the chief concern, there is a constant temptation, when the child is not gaining as rapidly as the mother thinks he should, to increase the food, regardless of conditions and beyond his requirements, usually with the result of seri-

ously disturbing the digestion. The best of all guides to increasing food is the child's demonstrated powers of digestion. If the child is not satisfied and digesting well it is usually safe to increase the food. But such increases should seldom be made more frequently than once in three days.

In increasing the quantity, it is not wise to add more than half an ounce to each feeding. During the early weeks both the quantity and the strength of the food should be increased every few days. It may be

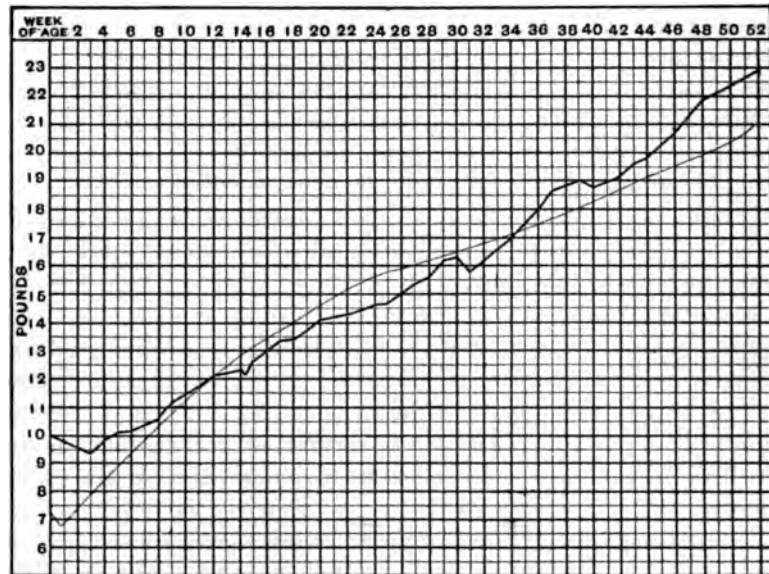


FIG. 30.—WEIGHT CURVE OF ARTIFICIALLY FED INFANT, SHOWING THE EFFECT OF BEGINNING WITH TOO HIGH PERCENTAGES. Robust child; digestion deranged when a few days old by beginning with fat 2 per cent, sugar 6 per cent, protein 0.75 per cent; food in two or three days was increased to fat 3 per cent, sugar 6 per cent, protein 1 per cent. A good deal of indigestion resulted, and the disturbance was such that it was eight weeks before the digestion became normal and the gain in weight regular; progress for the rest of the year satisfactory.

difficult to tell which of these it is best to do. It is well to alternate; thus, when the infant requires more food, first to increase the quantity; then, after a few days, if still unsatisfied, to increase the strength; the next time, to increase the quantity again, etc. In this way will be avoided the error into which mothers and nurses often fall, who adopt a single formula and keep on simply increasing the quantity indefinitely whenever the child is unsatisfied. I have frequently seen infants of two or three months taking as much as 7 or 8 ounces every two hours, and even then crying from hunger. After a daily total of 32 to 36 ounces is reached, as happens with most infants by the fourth month, the increase in the food should be chiefly in strength; for the same child at eight months will rarely require more than 40 to 45 ounces.

A caution is necessary against changing the formula too frequently. It is not possible to modify the milk in such a way as to relieve every trivial discomfort or disturbance an infant may have. Nurses are usually ready to ascribe every slight symptom to the food, particularly if they have strong opinions of their own upon the subject of feeding, and are not in full sympathy with the method employed. Very often the cause is outside of the food and even of the organs of digestion. Unless some very definite symptoms of indigestion, such as severe colic, vomiting, etc., are produced by the formula ordered, it is usually better to continue with it for at least two days, as it is hardly possible in a shorter time to determine what the child's digestive organs are capable of doing. For slight disturbances of a transient nature it is usually enough to dilute the food for a day or more; just before the bottle is given, one ounce or more of milk may be poured off and replaced by boiled water.

*Methods of increasing the Fat and Protein.*—To increase the fat and protein at the same time, one more ounce of the milk already employed is added in the 20-ounce mixture. In other words, one uses successively No. IV, V, VI, etc., of series A, B, or C, etc.

To raise the fat without raising the protein, one should use the same number of ounces, but employ a milk with a higher fat content; e. g., one is using No. VI, series D, with fat 1.20, protein 1.05. The fat is raised to 1.50, by using a 5-per-cent milk in place of a 4-per-cent milk; to 1.80, by using a 6-per-cent milk; to 2.10, by using a 7-per-cent milk.

To raise the protein without increasing the fat, a larger number of ounces in twenty are used, but of a milk with a lower fat content; e. g., one is using No. IV, series A, fat 1.40 per cent, protein 0.70 per cent. The protein is raised to 0.85 by using 5 ounces of series B; to 1.05, by using 6 ounces of series C; to 1.20, by using 7 ounces of series D, etc. Although the fat in this is not exactly the same the variation is so slight that it may be ignored.

*Conditions determining the choice of Milk Formulas.*—It has already been stated that with a healthy young infant one should begin with protein of not over 1 per cent and fat of 1.00 or 1.25 per cent. With formulas derived as these are, from 4- and 5-per-cent milk, respectively, very many infants can be successfully carried through the first year by merely increasing the number of ounces of the milk in the formula according to the gradations given in the table. Most healthy infants, however, with the percentage of protein which we wish to give, can take a higher percentage of fat than is given in formulas from 4- or 5-per-cent milk. It is therefore advantageous and, from a point of view of nutrition, it is economical, to give them a higher fat percentage, such as is obtained by using formulas derived from 6- or 7-per-cent milk. The fat percentage in the food as given should not, however, be raised above 4 per cent, and under most circumstances it is wise to stop somewhat



below this. When formulas containing the higher fats, such as those mentioned, cause vomiting, loss of appetite, or symptoms of intestinal indigestion, they should be immediately discontinued and formulas derived from milks of lower fat percentage substituted.

Children with feeble digestion or those suffering from disturbances of digestion should not be placed upon formulas derived from milk containing 7, 6, or 5 per cent of fat. Often with advantage they may be put upon formulas derived from milk containing even less fat than does whole milk. The special indications for such children will be considered more fully later.

**TO REDUCE MILK FORMULAS TO PERCENTAGES.**—In order to appreciate the composition of any milk formula which a patient may be taking it is necessary to reduce this to its approximate percentages. One who forms the habit of making such calculations soon finds it easy, and secures a basis for comparison with the percentages given as proper for the average normal child. A simple method of calculation is as follows: To determine the percentage of any constituent in the food, multiply its percentage in the original milk, cream, or top-milk by the number of ounces of each in the food, and divide by the total number of ounces of food prepared.<sup>1</sup>

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<sup>1</sup> A patient is taking a formula composed of cream 4 ounces, milk 16 ounces, milk sugar  $1\frac{1}{2}$  ounces, in a mixture containing 36 ounces. The cream is ordinary centrifugal cream, estimated to have 20 per cent fat; the milk is good average milk, estimated to have 4 per cent fat.

$$\begin{array}{rcl} 4 \times 20 = 80, & \text{which represents the fat in the cream} \\ 16 \times 4 = 64, & \text{" " " " " " milk} \\ \hline 144, & \text{" " " " " " total food} \end{array}$$

$$144 \div 36 \text{ (number of ounces of food)} = 4, \text{ the percentage of fat in the food.}$$

The protein is calculated in the same way. In the illustration we estimate the protein of 20 per cent cream at 3.05; in the whole milk, at 3.50 per cent.

$$\begin{array}{rcl} 4 \times 3.05 = 12.20, & \text{which represents the protein in the cream} \\ 16 \times 3.50 = 56.00, & \text{" " " " " " milk} \\ \hline 68.20, & \text{" " " " " " total food} \end{array}$$

$$68.20 \div 36 = 1.90, \text{ the percentage of protein in the total food.}$$

In a similar way, sugar is calculated. The sugar of a 20 per cent cream may be estimated at 3.90; in the milk, 4.50 per cent.

$$\begin{array}{rcl} 4 \times 3.90 = 15.60, & \text{which represents the sugar in the cream} \\ 16 \times 4.50 = 72.00, & \text{" " " " " " milk} \\ \hline 87.60, & \text{" " " " " " total food} \end{array}$$

$$87.60 \div 36 \text{ (number of ounces of food)} = 2.40, \text{ the percentage of sugar in the food before any is added.}$$

To add  $1\frac{1}{2}$  ounces to a 36-ounce mixture adds approximately 4 per cent of sugar; for 1.5 is 4 per cent of 36 [ $1.5 \div 36 = .04$ ].

The total sugar in the mixture therefore is  $2.40 + 4$ , or 6.40 per cent.

The formula contains therefore, approximately, 4 per cent of fat, 1.90 per cent of protein, 6.40 per cent of sugar.

**SPECIAL MODIFICATIONS REQUIRED BY PARTICULAR SYMPTOMS.—**

Most of the children for whom the physician's advice is sought in matters of feeding are not thriving, or, besides, are suffering from some evident symptoms of indigestion, and for these reasons changes in the food are required. In adapting milk for such cases one must rid his mind entirely of the notion that the food can be prescribed according to the child's age or even its weight, although both must be taken into account. The essential thing is the condition of the digestive organs, and unless this is carefully considered, failure is almost inevitable. To decide as to the proportions with which it is best to begin one must know, besides the age and weight, the nature and quantity of the food which has been taken, the appetite, the number and character of the stools, and also whether digestive symptoms are present, such as vomiting, flatulence, diarrhoea, colic, or constant discomfort. In any case the first prescription is somewhat in the nature of an experiment, but if the symptoms have been intelligently judged the experiment is likely to prove successful.

Even with infants who are properly fed there are few whose digestion remains perfectly normal throughout the entire first year. Changes in the food are therefore necessary from time to time, even in the most healthy, to meet special symptoms which may arise. Many of these are due to disturbances of a minor character, but are none the less important, as they may lead to serious consequences when not immediately recognised and properly treated.

*Vomiting.*—The common causes of habitual vomiting referable to the food are: too high fat or too high sugar, especially if the sugar is either maltose or cane sugar, too frequent feedings and too much food at one time. Frequent vomiting or regurgitation, often one or two hours after feeding, of curdled milk or of a sour, watery fluid, is usually an indication that the proportion of fat is too high. Sometimes it is the sugar that is in excess, and sometimes both fat and sugar are at fault. The first indication is to reduce the fat. Formulas from top-milk or milk and cream should not be used, but rather formulas from whole milk; and if the vomiting is frequent, formulas from skimmed milk are advisable for temporary use, afterward those from whole milk. If a reduction of the fat does not give relief the sugar should also be reduced and neither maltose nor cane sugar should be used. Other changes which are sometimes helpful are to use twice the usual amount of lime-water, making this 10 per cent, or 2 ounces in each 20-ounce mixture.

An infant who vomits often should not be fed at shorter intervals than three hours, even if only two or three weeks old. If considerable quantities are ejected almost immediately after feeding, it is usually because too much food has been given. Other causes must be considered

also—the food may be too rapidly taken, the child may be moved about too much, the abdominal band may be too tight, etc.

*Constipation.*—The principal causes of constipation referable to the food are too low total solids and too low fat. Habit and general training are also important factors. Sterilisation, and to a slight degree pasteurisation, cause milk to be somewhat constipating. During the first few weeks, if the percentages are low, there is often a species of constipation present which is simply the result of the low total solids in the food given. The bowels usually move every day, sometimes even twice a day; but the stools are often small and rather dry. Unless there is manifest discomfort on the part of the child, such a condition may be disregarded, especially if the odour and colour of the discharges are nearly normal. As the proportions of all the elements of the food are gradually increased along the general lines previously indicated, this form of constipation passes away. Mothers and physicians often expect that the bottle-fed infant will have during its first month or two the two or three large stools daily to which they have been accustomed with healthy breast-fed infants. But finding instead only one movement a day, and that small and sometimes dry, they resort to laxatives or enemata, and by their use really cause much of the trouble they are seeking to remove.

The low fat is often the explanation of the constipation seen when infants are fed upon formulas derived from whole milk. If such is the case relief may sometimes be afforded by changing to formulas made from milk containing 6 or 7 per cent fat. The increase in the fat to overcome constipation can only be carried up to a certain point; this is generally 2.5 or 3 per cent for a young infant and 3.5 or 4 per cent for one who is older. If the fat is raised beyond this other disturbances of digestion, particularly vomiting, are likely to result, and sometimes there may even be an increase in the constipation. Some other means of overcoming the constipation should be resorted to.

The substitution of the milk of magnesia for lime-water in milk modifications is often of service. It may be continued for several months without harm. One teaspoonful added to the total food for the day is usually sufficient; this amount may be slightly increased or lessened according to the effect produced.

Milk sugar is somewhat laxative and the raising of the proportion of this ingredient as high as 7 per cent, if a smaller proportion is being used, will often be all that is needed. Maltose is much more laxative in its effect, and may be substituted wholly or in part for milk sugar. Its use will be more fully discussed later. It should not be given if there is vomiting.

*Colic and Flatulence.*—The habitual colic of early infancy may occur with any form of intestinal indigestion; its causes therefore are varied.

Colic and flatulence are especially common in infants who suffer from constipation. Excessive flatulence may occur also when cereal gruels are added to the milk of young infants, particularly if the amount is considerable and if the cereal is insufficiently cooked. If symptoms are severe a reduction in all the elements of the food may be necessary.

*"Curds" in the Stools.*—The undigested masses appearing in the stools of infants taking milk are usually spoken of as "curds." These may be small, soft, and white, and may make up a large part of the stool. An excess of mucus is usually present. Such masses are composed almost entirely of fat. There are also seen, but much less frequently, larger, smooth, hard masses of a yellowish-brown colour, but white on section. They are generally present in small numbers in a stool the rest of which may be quite normal. These are composed chiefly of protein, usually with an envelope of fat. Curds of the first variety, if numerous, call for a considerable reduction in the fat percentage. The smooth, hard curds, if numerous and persistent, should lead one to reduce the protein at least temporarily.

*Loose, Green, or Yellowish-green Stools of a Sour Odour.*—These are sometimes due to too high a percentage of sugar, especially lactose, often to an excess of fat. The number of stools is usually from two to five daily. In appearance the stools resemble thin scrambled eggs. Stools such as those described are often seen in nursing infants as well as in those artificially fed, and the condition is not incompatible with steady and regular gain in weight. After it has persisted any length of time mucus is regularly present.

*Large, Dry, Clayey Stools.*—These are often smooth, and are generally due to an excess of fat. They have usually a peculiarly foul odour, owing to the presence of fatty acids.

*No Gain in Weight without evident Symptoms of Indigestion.*—This is sometimes due to too weak food, all the percentages being too low, the child usually manifesting signs of hunger. Occasionally it is due to the fact that all the percentages, particularly that of the fat, are too high. In the latter case it frequently happens that the appetite is much reduced, so that the infant takes perhaps less than half his usual allowance. A considerable reduction in the fat and an increase in the sugar, particularly the addition of maltose, will often lead to immediate improvement. The amount used should at first be small, not more than two or three drachms to the day's food, and gradually increased to half an ounce or an ounce a day. Too frequent feedings and the practice of constantly coaxing the infant to take more food, often produce the same aversion to food. It is much better to offer food at three or four hour intervals and take away the bottle as soon as the child shows that he does not want more.

Modifications in the food to meet the indications afforded by more

serious conditions than those here described are considered in the later pages devoted to Difficult Cases of Feeding.

**THE APPARATUS REQUIRED FOR THE PREPARATION OF MILK AT HOME.**—This includes a glass graduate, a glass or agate funnel, a cream dipper, a pitcher for mixing food, feeding-bottles, a tall cup for warming the food, a small ice-box, and a steriliser. Other articles needed are lime-water, milk sugar, rubber nipples, absorbent cotton, bottle-brushes, borax or boric acid, bicarbonate of soda, and an alcohol lamp, or better, if gas is available, a Bunsen burner. The best style of bottle is that which can be most readily cleaned. The graduated cylindrical bottles with wide mouths are to be preferred. The best nipples are those of plain black rubber, which slip over the neck of the bottle, and are not so thick as to prevent their being turned inside out for cleansing. Those with a long rubber tube going to the bottom of the bottle should not be used. In many places their use is prohibited by law. The hole in the nipple should be large enough for the milk to drop rapidly when the bottle is inverted, but not so large that it will run in a stream. New nipples should be boiled; but the daily boiling of nipples is unnecessary. It soon makes them so soft as to be useless. They should be rinsed in cold water immediately after using and washed daily in soap and water. When not in use, nipples should be kept covered in a solution of borax or boric acid. Bottles should first be rinsed with cold water, then washed with hot soap-suds and a bottle-brush. When not in use they should stand full of water. Before the milk is put into them they should again be placed in boiling water for a few minutes.

**DIRECTIONS FOR PREPARING THE FOOD.**—All the food needed for twenty-four hours should be prepared at one time. The first thing to be decided is the formula to be used; next, the quantity of food for twenty-four hours with the number of feedings into which it is to be divided.

Let us suppose, for example, that the child to be fed is a normal three-months-old infant weighing twelve pounds. Referring to the table of formulas previously given, we first decide upon the percentage of protein to be used; 1.20 or 1.40 per cent would seem appropriate. By referring to the figures in the column on the extreme left we see that 1.40 per cent of protein is obtained by using 8 ounces in 20 of any of the various milks. We may obtain 2.80 per cent of fat if we start with 7-per-cent milk; 2.40 per cent of fat if we start with 6-per-cent milk; 2 per cent of fat if we start with 5-per-cent milk, etc. It is probable that such a child as that mentioned could take 2.80 per cent of fat without difficulty. Instead, however, of using this at the outset, a safer plan would be to start with 2 per cent, and later, if this was well borne, raise the proportion gradually to 2.40 and 2.80 per cent. A mixture having 2 per cent of fat and 1.40 per cent of protein is, as seen from the table, obtained by diluting 5-per-cent milk.

The proper amount of sugar would be 6 per cent. The milk having 1.40 per cent of protein has but 1.80 per cent of sugar. It is therefore necessary to add 4.20 per cent, to bring the proportion up to the desired amount. Since one even tablespoonful in 20 ounces adds 1.75 per cent of sugar, about two and a half tablespoonfuls in 20 will be needed.

The formulas would therefore be:

|                       | For 20 ounces.     | For 30 ounces.     | For 40 ounces.    |
|-----------------------|--------------------|--------------------|-------------------|
| 5-per-cent milk ..... | 8 oz.              | 12 oz.             | 16 oz.            |
| Milk sugar .....      | 2½ even tablesp'ls | 3½ even tablesp'ls | 5 even tablesp'ls |
| Lime water .....      | 1 oz.              | 1½ oz.             | 2 oz.             |
| Water .....           | 11 oz.             | 16½ oz.            | 22 oz.            |
|                       | 20 oz.             | 30 oz.             | 40 oz.            |

Such a child as the one assumed would probably take seven feedings of 5 ounces each.<sup>1</sup> It would be well to prepare 40 ounces of food and have one extra bottle on hand in case of accident.

The milk sugar should be dissolved in boiled water, which is then mixed with the milk in a pitcher and the lime-water added. The food is now divided into the seven bottles, which are stoppered with cotton. They are placed at once in an ice chest, or first sterilised, then cooled, and afterward placed upon ice.

**DIRECTIONS FOR FEEDING.**—The food should be warmed to about 100° F. before feeding, best by placing the bottle in a tall pitcher or cup filled with hot water, not by pouring the food from the bottle into a saucepan. The temperature of the food may be tested by the nurse with a thermometer, or by pouring a few drops upon the front of the wrist; it should feel warm, but not hot. The nurse should never take the nipple of the bottle into her own mouth. A bottle should not be warmed over for a second feeding. A child should not be more than twenty minutes in taking its food, and should not sleep with the nipple of the bottle in his mouth. It is preferable to have a young infant held while taking his bottle. If this is not done, the bottle should at least be held in such a position that the neck of the bottle is kept full, so that the child gets milk, and not air. It is even more necessary than in breast-feeding that rules as to frequency and regularity of meals be observed.

**The Use of other Food than Milk during the First Year.**—In the discussion up to this point nothing but the elements of milk has been considered. Upon these alone the average healthy infant is best nourished

<sup>1</sup> Calculating the calories in the food offered, by the use of the table of caloric values already given, it will be found that the 35 ounces of food will furnish about 510 calories, which will represent about 95 calories per kilo. of body weight

for the first four or five months. The use of the various cereals as an addition to the milk for young infants is a useful measure for some infants, but not desirable for all. The early use of much farinaceous food often results in serious harm.

For the average healthy infant it is desirable to begin with farinaceous food in some form by the fifth or sixth month. By this time the power of digesting starch is sufficiently strong for the infant to receive some of its carbohydrates in this form, instead of all of it in the form of sugar, as has been previously the case. As starch is added, the sugar may be gradually reduced. The form of starch used may be a gruel made of wheat, barley, oatmeal, or arrowroot. This will take the place of part or all of the boiled water in the preparation of the food. It is thus given with each of the feedings. By the eleventh or twelfth month the quantity of the cereal may be considerably increased.

The only other things to be advised during the first year are beef juice and the juice of some fresh fruit. Beef juice may be begun in the tenth or eleventh month, earlier with anæmic children; at first not more than two teaspoonfuls daily, later the amount may be increased. The best fruit juice is that of the orange, which should be fresh and sweet. It may with advantage be given to most infants ten months old, and to many when seven or eight months old. Beginning with half an ounce, the quantity may be gradually increased to two ounces, given preferably about one hour before the second milk-feeding.

**The Tolerance of Healthy Infants for the Different Food Elements.—**

In the foregoing pages we have indicated the percentages which, in our experience, have been shown in the majority of instances to be the best for feeding healthy infants. However, Nature will often tolerate very wide variations from what is best. The desire for a rapid increase in weight often leads to an increase of the fat in the food much beyond the limits which are usually considered safe. There are some children of vigorous constitution and strong digestion, living under good surroundings, who tolerate this for a long time; some may even go through infancy to a period of mixed diet without any visible disturbance, and appear to thrive exceedingly well. There are others who bear such high fat proportions for a considerable time and then show serious disturbances. They thrive so long as all the other conditions are perfect; but the slightest deviation from these conditions, as, for example, some mild intercurrent illness, tonsillitis, bronchitis, etc., possibly so slight a thing as dentition, may bring about an acute condition which may be of a most alarming character. Most frequently it is the advent of very hot weather which is the occasion of the breakdown. There are others who are upset almost from the beginning if high proportions of fat are used. Still others gradually develop subacute or chronic disturbances of digestion and nutrition which may last for months. What is true of the

use of excessive amounts of fat is true to a less degree of the sugar and very rarely of protein also. One should be very cautious, therefore, in inferring that, because a few exceptionally strong infants thrive on unusual proportions or excessive amounts of some one of the food elements, this is to be taken as a guide in feeding the average child.

#### FEEDING IN DIFFICULT CASES.

There are included under this head, infants who, owing to feeble digestion or individual peculiarities, do not thrive, even from the outset, upon the usual milk modifications, although they may be used in-

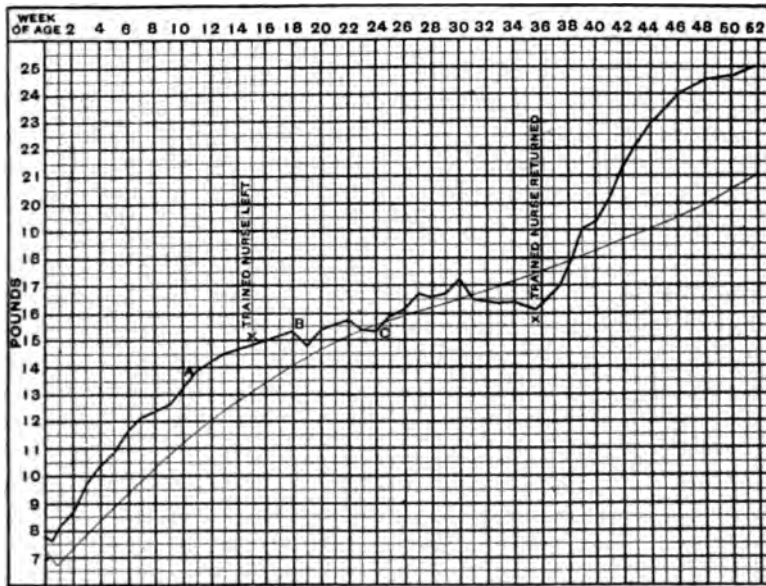


FIG. 31.—WEIGHT CHART SHOWING THE EFFECT OF INTELLIGENT CARE. Maternal nursing in the beginning; A, began part feeding; B, attack of indigestion; C, weaned entirely. The departure and return of the trained nurse are indicated upon the chart. In the interval there was constant indigestion for which no sufficient explanation could be found in the food. Subsequently this was discovered to be due to the carelessness and neglect of the nurse. Immediate improvement on the return of the trained nurse without any important change in the food. It will be noticed that during the four and one-half months of the trained nurse's absence the net gain in weight was only 1 pound 3 ounces.

telligently, and a much larger group who have prolonged disturbances of digestion, the result of previous improper methods of feeding. In the aggregate the number of children included in these two groups is large, and few cases in the practice of the physician cause him more trouble or anxiety. Even one of large experience often finds himself baffled by the problems which individual cases present. The difficulties are



greatest in early infancy, in cities, in institutions, in hot weather, and they are further increased by the existence of constitutional debility, and where the trouble is of long standing. That chronic indigestion in a young infant is a serious thing is often not appreciated. The mother is apt to think the problem one easy of solution; she only wants to be told what to feed her baby, imagining that a single food prescription should set the child right at once. The physician, too, sometimes regards the condition lightly because these infants do not seem really ill; he therefore considers the subject hardly important enough for his careful, continuous attention. What I wish to emphasise is that these cases are serious, that they are difficult, that in most of them nothing can be accomplished without close and continuous personal observation, that they do not tend to right themselves, and that infants' lives are often sacrificed as a result of bad management.

While these infants present great variety in their symptoms, and must be carefully individualised in their management, there are some general principles applicable to all. One should begin by obtaining a careful history of what has been previously tried, in order to get all possible information respecting the type of indigestion which the child presents. These previous efforts in feeding should be studied with great minuteness; the different changes made and the effect of each one upon the principal symptoms, the vomiting, the stools, and the child's weight should be considered. With a good history obtained from an intelligent mother or nurse one can often at once determine where mistakes have been made, and in many cases the same mistake has been repeated with each change of food.

A thorough investigation into the nursery routine should be made to ascertain not only what has been tried, but how it has been tried. It is frequently found that the failure is due not to any fault with the food prescribed, but to other conditions. The food may be improperly prepared or given—e. g., it may be cold or given too rapidly; the bottles or nipples may be dirty; the proper quantities and intervals not observed, etc. Another factor of importance is the environment as affecting the nervous system of the infant. Among the well-to-do this may be the chief trouble. The constant or frequent excitement by visitors, or playing with a child by parents or nurses, may result not only in lack of sleep, but in disturbances of digestion, often in habitual vomiting, though the food itself is proper. Under such circumstances the removal of the child from its surroundings or placing it in charge of a competent nurse will often cause an immediate and marked improvement without any change in the food. Another cause of disturbance is the habitual use of the "pacifier," something frequently resorted to in these cases, but which should under no circumstances be tolerated. Success in treatment will depend largely upon how accurately one is able to discover the

essential cause or causes of trouble and the nature of the disorder of digestion in the case under treatment. Without such knowledge all is haphazard experimentation.

In dealing with these cases drugs are of little assistance; in most cases they are better omitted altogether.

In carrying out any line of treatment little can be accomplished without continuous observation at fairly frequent intervals on the part of the physician and the co-operation of an intelligent mother or nurse. Particular attention should be paid to the stools, which the physician should see for himself, to the presence of colic or flatulence, vomiting, the appetite, and the body weight. A daily record is of great assistance. The weight, though important, is not the only guide as to progress. It should be taken regularly in order that a steady loss may not go on unnoted; but the first signs of improvement are usually observed in other symptoms—the child is more comfortable, sleeps better, and suffers less from his special disturbances of digestion.

Generally speaking, the intervals between feedings should be longer than for infants with good digestion. It is never wise to make them less than three hours for young infants, or less than four hours for those who have passed the eighth or ninth month.

Regarding the effect upon the digestion of concentration of the food (i. e., a large quantity of a weak food, or a small quantity of a strong food), different results are seen with different children. The usual tendency when an infant suffers from indigestion is to dilute the food, and in some cases this is perfectly proper; but to continue increasing the dilution because the patient does not do well may be the very worst treatment. This may do harm by causing too much dilution of the digestive fluids. Small feedings, not weak food, are what benefit some of these children most, the balance of the daily amount of water needed by the child being given between the feedings.

In very troublesome, protracted cases minor variations in the composition of the food or slight changes in the plan of feeding rarely accomplish much. Radical changes are usually necessary. If small feedings at short intervals have failed, one may succeed with larger feedings and much longer intervals. If very dilute food in large quantities has failed, improvement may follow much smaller feedings and a much stronger food. For similar reasons the most brilliant results are often obtained from as complete a change in the diet as possible. An infant who has been long on farinaceous foods is most likely to improve when these are stopped entirely and suitable percentages of cow's milk given. One whose digestion has become seriously deranged while taking milk, and whose symptoms have continued in spite of many intelligent variations in the food, is sometimes helped by nothing so much as temporarily withdrawing all milk.

**Clinical Types.**—The greater number of these cases may be divided into three groups: (1) Those whose chief symptom is habitual vomiting, or regurgitation of food; (2) those with intestinal symptoms, most frequently with loose stools; (3) those without any marked symptoms of indigestion, yet whose weight is much below the average, who do not gain on weak food and are upset if stronger food is used. They have feeble digestion rather than indigestion.

The first group, the cases with vomiting, are the least difficult of the three to control. The causes which produce this are usually more obvious and more easily removed. Altogether the most frequent cause of symptoms of this kind is the use of too high fats of cow's milk. The proportions used may not be improper for normal children, but they are excessive for the particular patient. A less frequent cause is high carbohydrates, especially foods containing maltose, sometimes cane sugar. Some of these children vomit only occasionally and in large quantities; but the frequent regurgitation of undigested food in small quantities, often several times after each feeding, is more characteristic. After a time the vomitus invariably contains more or less mucus. Vomiting of this type is to be sharply distinguished from that which occurs only immediately after feeding from overfilling of the stomach. The symptoms also should not be confused with those of pyloric stenosis. Often the early mistakes in feeding are not gross ones, but the gastric disturbance becomes established because suitable changes in the food are not at once instituted.

In the second group, the cases with intestinal symptoms are the most difficult to control. Usually, if not actual diarrhœa, there are at least frequent stools, from three to six a day of almost every conceivable colour and variety, and large in proportion to the quantity of food taken. Mucus is almost always present; in the more severe cases and in those of long standing the quantity may be excessive. Exceptionally the children suffer from constipation. Such cases, however, are generally easier to manage, as there is not the same intolerance of food; but these patients may suffer from abdominal distention, flatulence, and colic. The most important element in the food which produces the first disturbance in these intestinal cases is excessive fat; frequently, also, it is due to excessive carbohydrates, particularly cane sugar or maltose, and sometimes to starchy foods. Very soon, in severe cases, all the elements of the food, but especially fat and sugar, are badly borne. After the condition has existed for some time it may be impossible to determine which of the food elements is really doing the most harm.

In the third group, the cases with feeble digestion, mistakes in feeding are less prominent as causes. Much more important is the feeble constitution. This may be the result of prematurity or of congenital feebleness. The most striking feature of these patients is the extreme

sensitiveness of their digestive organs to even the smallest deviation from the best methods of feeding. The slightest mistake may result in a serious upset, and it may be several weeks before the child is as well as before. Often under the best surroundings, and when fed with the greatest care and intelligence, such infants do not thrive.

**Management.**—Passing now to the treatment of these different conditions we find that, so far as the elements of cow's milk are concerned, the greatest difficulty is with the fat; this applies particularly to the first two groups. Next to the fat, the most trouble is with the carbohydrates. Of the sugars, maltose is more likely to disagree than cane sugar or starchy foods, while milk sugar is by most children the most easily borne. That protein causes trouble also, and how and when it does so, is much less evident and lacks conclusive proof. This appears to be true at times in very young infants.

With these points in mind it will be evident that, for the class of patients under consideration, top-milk or milk and cream mixtures are not admissible. For nearly all of them the fat must be even lower than in formulas made from whole milk. According to the severity of the symptoms, there should be employed dilutions of 3-per-cent, 2-per-cent, or 1-per-cent milk, and in extreme cases even fat-free milk. It is also a principle of wide application that cases with predominant gastric or intestinal symptoms tolerate maltose badly, and in most cases cane sugar also. Some marked cases may be unable to tolerate more than a small amount of milk sugar.

At the very outset it should be clearly borne in mind that notwithstanding the fact that these patients are much below normal weight, and often losing steadily, the treatment should be directed first of all to allaying the most marked symptoms of indigestion. Until these are relieved no permanent improvement can be expected. For the time being the weight must be disregarded. No time should be lost in attempting to correct the digestive symptoms by the use of drugs or the administration of digestive ferments. Our resources for controlling these cases are chiefly variations in the food.

The milk modifications which are suitable in different cases are: (1) Formulas from partially or completely skimmed milk; (2) buttermilk and other fermented milks; (3) protein milk (*Eiweiss-milch* of Finkelstein); (4) condensed milk. (Their preparation has already been described in the chapter on Cow's Milk.) (5) Wet-nursing; (6) substitutes for milk.

The variations which may be obtained from skimmed milk are sufficient for the relief of a large number of the cases met with, particularly those with gastric symptoms. Such an one, three or four months old, with symptoms of moderate severity would probably take 1 to 1.50 per cent of protein, but not more than 0.6 to 0.8 per cent of fat. Such

formulas can be derived, as may be seen from the table, from 2-per-cent milk. The sugar should seldom be higher than 5 per cent. With improvement in the symptoms the proportion of all the ingredients may be gradually, but very slowly, raised; it will usually be months, however, before such a patient can take as much as 2 per cent of fat. A similar plan of treatment will sometimes succeed when the symptoms are intestinal, but in such cases one must be cautious in the use of sugar.

Buttermilk and the other fermented milks are indicated particularly in cases with intestinal symptoms. Their advantages over skimmed milk are that they not only have a lower fat content, but a lower sugar as well, and contain some lactic acid. They succeed in a certain number of cases that do not respond to skimmed milk. It is seldom necessary to dilute them more than with an equal amount of water.

Protein milk is indicated in cases with intestinal symptoms where it is desired to reduce the sugar as much as possible, but still retain a considerable proportion of the fat. It is not necessary to dilute this with more than an equal volume of water, and it may be given from this dilution up to full strength. In many cases with marked intestinal symptoms it is more efficacious than any of the other milk modifications.

Condensed milk is the direct opposite of protein milk in that both the fat and the protein are low while the carbohydrate is high and chiefly cane sugar. It is often difficult by symptoms alone to determine the precise indications for using condensed milk in cases with intestinal symptoms, but the fact remains that in certain cases it has undoubted value. It may be diluted with plain water, but often its effect is better if the diluent is barley water.

For the first and second groups of cases the milk of a good wet-nurse is seldom the best food. Its high fat content will usually aggravate the vomiting and increase the diarrhoea. Its use should therefore be deferred until the digestive symptoms are under control. At a later stage it may be invaluable for increasing weight. For the third group, the children with feeble digestion, wet-nursing is unquestionably the most successful method of treatment.

The stopping of all milk is at times a useful procedure. However, this should be done only for a limited time, a few days to a week at most. The proprietary foods, under such conditions, seldom prove valuable and often do much harm whether used alone or added to milk. Those children who have trouble with fats and sugars are sometimes enabled to take a sufficient amount of farinaceous food to maintain the body weight for some time. For such purposes a barley, wheat or oat gruel may be used, but it should be made strong—two tablespoonfuls of flour to a pint, occasionally even stronger than this. A substitute sometimes useful is a gruel made from the soy bean. This is high in vegetable nitrogen and low in starch while it contains considerable fat. It

may be continued as the sole diet for a short time during periods of marked disturbance. To any of the above substitutes milk should be added in small quantities as soon as possible; at first either fat-free milk or skimmed milk should be employed.

All the above resources, except feeding by a wet-nurse, are to be looked upon as methods of relieving digestive disturbances, not as permanent foods. When the symptoms have disappeared and there is no longer vomiting and the stools have approached the normal, other food stuffs may be employed to increase weight. The most valuable one of these is maltose. Maltose<sup>1</sup> has the advantage over all sugars in point of easy assimilation. It has also the disadvantage in that it breaks down more readily than do other sugars. It should not therefore be employed so long as either gastric or intestinal symptoms are present. The directions upon the package should not be followed, but the amount added should at first be small, i. e., one teaspoonful of a maltose solution or malt-soup extract to the daily food. This may be increased every few days until the total amount is from six to eight teaspoonfuls daily. If it causes vomiting or too frequent stools it should be omitted.

Another valuable food stuff is olive oil. It is a form of fat which can at times be tolerated when the fat of cow's milk habitually disagrees. I have used olive oil in cases of this kind for the past two years with, in many instances, most striking benefit. Some children who are unable to take as much as one per cent of the fat of cow's milk bear olive oil without difficulty. The amount used at first should be small, not more than one-half teaspoonful three times a day. It may be gradually increased until one-half teaspoonful is given directly after each feeding, six or seven times a day. The maximum amount to be used for infants of the first year should seldom be over half an ounce daily. Only exceptionally when used in this way does it cause diarrhoea and still less frequently does it excite vomiting. It is therefore of value as a form of fat which may be given to infants whose greatest difficulty in digestion is their inability to tolerate milk fat. The chief means by which weight can be increased in children suffering from malnutrition is therefore through the addition of carbohydrates, next by the addition of fat, but neither of these is to be employed in any considerable quantity until the marked symptoms of indigestion have been controlled. As a means

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<sup>1</sup> Many malt preparations sold in the market also contain diastase, which is not desirable for use under the conditions here considered. Loefflund's and Borchardt's "malt-soup extracts" are reliable but expensive. Also reliable and much more moderate in price are the "neutral maltose" of the Maltzyme Co., Brooklyn, and the "malt syrup" of the Freihofer Bakery Co., Philadelphia. Both these preparations are somewhat acid. To the former, five grains and to the latter ten grains of potassium carbonate should be added to each ounce of the malt before it is used in the food. All of the preparations mentioned contain from 65 to 85 per cent of carbohydrates, about two-thirds of which is maltose and the balance chiefly dextrin.

of allaying such symptoms nothing compares with the various modifications of cow's milk above described.

The use of formulas made from whey has already been referred to in the chapter on laboratory feeding. Whey mixtures are indicated whenever there is especial difficulty in digesting the casein of cow's milk. It may be hard to tell by symptoms when this is the case. It is more often true of very young infants than in those who have reached the age of three or four months. Such infants are frequently constipated and suffer in consequence from flatulence and colic. Plain whey may be used, or the fat may be raised by adding a small proportion of 7-per-cent milk (one ounce in twenty), or the carbohydrates may be increased by the addition of maltose. Some patients are helped very much by whey mixtures; very many are not helped at all for the reason that the trouble with such patients is not in digesting casein.

What has just been said of whey applies also to the use of peptonised milk. It is employed too frequently, and is apt to be continued too long, and but very few of the troublesome feeding problems are solved by its use. Citrate of soda has been added to milk, usually in the proportion of one grain to the ounce of food, with the belief that a delay in the coagulation of casein in the stomach which this brings about, is a desirable result. Neither on theoretical nor practical grounds can I see any reason for its use. Although I have tried it extensively I can not say that I have ever seen any marked benefit from it.

The disturbances of nutrition with which the difficult feeding just described is associated, have been regarded by Finkelstein from a point of view somewhat different from the usual one. He groups infants with nutritional disturbances not so much according to the character of their stools or their previous digestive symptoms as by the way in which they react to food. He thus attempts to classify them on the basis of their functional capacity.

A child with normal digestion and perfect nutrition has a temperature which fluctuates within narrow limits, and he responds to a proper increase in food by a gain in weight. On the other hand, the response of the abnormal child to food is something quite different and varies according to the degree of his disturbance.

In the mildest grade of nutritional disorder, that of disturbed equilibrium (*Bilanzstörung*), with sufficient food there is no regular gain in weight; but the weight fluctuates for a considerable period until more serious symptoms develop or until an adjustment is reached. The stools may appear nearly normal but the infant's tolerance of food is considerably reduced. The temperature fluctuations also are wider than are seen in health.

If the condition is not relieved symptoms more definitely related to the digestive tract supervene, usually diarrhoea (*Dyspepsic*). The stools

are thin, green, and contain mucus. The loss in weight for some time is not marked, but other symptoms are more severe and from time to time there is a moderate elevation of temperature. In this stage the child's tolerance of food is still further reduced.

In the third degree of disturbance (*Dekomposition*) which follows after a longer or shorter period there is rapid and marked loss in weight. The temperature is usually below normal; the pulse, slow; the respiration, often irregular, and food tolerance falls to a minimum. The character of the stools depends much upon the diet. When the food is greatly reduced the stools may appear normal, but any increase in food is followed by bad stools. It will thus be seen that these three groups of Finkelstein represent the usual types seen, viz., slight, moderate, and severe nutritional disturbances due to improper feeding.

His chief contribution is in the emphasis laid upon the fact that under certain conditions the food elements, even though not in large amount, may be injurious. They may themselves produce most definite and severe symptoms which are in no way dependent upon bacterial infection.

Finkelstein's fourth stage (*Intoxication*) indicates a complete breakdown of all the processes of nutrition. This is discussed in a succeeding chapter.

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## CHAPTER IV.

### FEEDING AFTER THE FIRST YEAR.

#### HEALTHY INFANTS DURING THE SECOND YEAR.

THE physician should not relax his vigilance in the feeding of a child after the first year has passed. The ideas of the laity in regard to what a child can digest after it has outgrown an exclusive milk diet, are very erroneous. The majority of infants are given solid food too early, in too large quantities, and improperly prepared. Most of the attacks of indigestion during the second year are directly traceable to gross dietetic errors. The diet of a healthy child during the second year should consist mainly of milk, and some farinaceous food with bread, a small amount of animal food—beef or mutton, beef juice, eggs—and fruit juice or cooked fruit.

Milk should be the basis of the diet. The popular idea that there are many children who can not take milk is an erroneous one; the real trouble usually is that they will not take it because other food pleases the palate better, and they are allowed to have their own way in this as in other things. It is of the utmost importance that the transition from a purely fluid diet to one of solid food should be made very slowly, and that the habit of drinking milk should not be discontinued.



During the second year with average milk and average infants very little modification of the milk is required. The addition of milk sugar is unnecessary, since the child is now able to take a considerable part of his carbohydrates in the form of starch. If the milk is very rich, such as that from a Jersey herd, it should be diluted with at least one-fourth water. In hot weather a still greater dilution may be necessary.

*Weaning from the Bottle.*—This should always be begun by the thirteenth month; by the fifteenth month an infant should take all his milk from a cup, except possibly the 10 P.M. feeding, when the bottle may be allowed for the sake of convenience. Early weaning from the bottle is a matter of no small importance. When the bottle is allowed to older children the disposition to overfeeding especially during the summer may be very great. Again there are many children with the "bottle-habit" so developed that throughout childhood, although at any time they will take milk from the bottle, they can never be induced to take it any other way.

**From Twelve to Fourteen Months.**—The daily schedule at this period should be about as follows:

- 6.30 A.M. Milk, six to eight ounces; diluted with barley or oat gruel, two to three ounces.
- 9 A.M. Orange juice, one to two ounces.
- 10 A.M. Same as at 6.30 A.M.
- 2 P.M. Beef juice, one to two ounces;  
or, the white of one egg, slightly cooked; later, the entire egg;  
or, mutton or chicken broth, four to six ounces.  
Milk and gruel in proportions above given, four to six ounces.
- 6 P.M. Same as at 6.30 A.M.
- 10 P.M. Same as at 6.30 A.M.

In preparing the food, the milk and the gruel are simply mixed together while the latter is warm, and salt and a very small quantity of cane sugar added to make it palatable. It is then divided into as many feedings as are required for the day, each one being placed in a separate bottle. As to handling the bottles and pasteurising or sterilising, the same rules apply as during the first year.

**From Fourteen to Eighteen Months.**—The diet may be increased by the addition of more solid food. The average child will take:

- 6.30 A.M. Milk, warmed, eight to ten ounces.
- 9 A.M. Fruit juice, one to three ounces.
- 10 A.M. Cereal: one, later two or three, tablespoonfuls of oatmeal, hominy, or wheaten grits, cooked for at least three hours; for the first month or two this should be strained; upon the cereal from one to two ounces of thin cream, or milk and cream, with plenty of salt, but without sugar.  
Crisp dry toast, one piece; or, unsweetened zwieback;  
or, one Huntley and Palmer breakfast biscuit.  
Milk, warmed, six to eight ounces.

- 2 P.M. Beef juice, one to two ounces; and one egg (soft-boiled, poached, or cod-dled); and boiled rice, one tablespoonful, cooked four hours; or, broth (mutton or chicken), four ounces; one or two breakfast biscuits, or swieback; and (if most of the teeth are present) rare scraped meat, at first one teaspoonful, gradually increasing to one tablespoonful; milk, four to six ounces, if desired.
- 6 P.M. Cereal: two tablespoonfuls of farina, cream of wheat, or arrowroot, cooked for at least one hour, with milk, plenty of salt, but without sugar; or, bread and milk or milk toast.
- Milk, warmed, eight to ten ounces.
- 10 P.M. Milk, warmed, eight to ten ounces, which may be given from a bottle.

**From Eighteen Months to Two Years.**—The amount of solid food may be somewhat increased. The number of the meals should be the same as for the preceding period. In addition, cooked fruits, such as the pulp of stewed prunes or baked apple, strained, may be given at the mid-day meal. It is generally best not to give fruits and milk at the same meal. Nothing but water should be given between meals. Potato is best deferred until the child is nearly two years old, and other vegetables still longer.

#### DIFFICULT CASES DURING THE SECOND YEAR.

The number of children whose nutrition is a matter of difficulty during the second year is much smaller than during the first year; yet the difficulties may be just as great. Some of these are infants who have been very delicate from birth, and carried through the first year only by the greatest effort. Others are healthy at birth, but their digestion has been badly deranged in consequence of improper feeding. Still others did well until they were weaned. The conditions may be the result of a severe attack of acute disease of the stomach or intestines during the first year. Other important causes are the early use of solid food and the too exclusive use of farinaceous foods of all varieties.

Whatever the special cause of the condition, cases of chronic indigestion in the second year are to be managed along the same general lines as have already been laid down for those under one year. Usually the first thing to be done is to stop all solid food except possibly rare scraped beef. Starches must be greatly reduced or prohibited altogether. The milk should be modified as for healthy infants who are much younger than the patient under treatment. The daily quantity should generally be somewhat larger than for a young, healthy infant taking food of the same strength. The regular intervals of feeding should never be shorter than three hours, and usually intervals of four hours are to be preferred. A safeguard against overfeeding or underfeeding these patients is the determination of the caloric value of the food given.

Striking improvement often follows the administration of rare scraped meat, especially to those who are over eighteen months old. From one

to two ounces may be given daily. Generally the protein in the food has previously been deficient. Many of these children digest meat when given in this way better than they do milk. Beef juice and the whites of eggs, partially cooked, may also be given.

Fruits should be used with great caution. As it is with the starches that great difficulty is usually experienced, the carbohydrates should be administered chiefly in the form of milk sugar or some of the preparations of maltose.

When the child is once well started and gaining steadily, the food may be gradually increased, until the diet recommended for healthy infants of the same age is reached. All changes must be made very gradually, and it should never be forgotten that there is a constant disposition on the part of all mothers and nurses to overfeed these children.

#### FEEDING FROM THE THIRD TO THE SIXTH YEAR.

**Articles Allowed.**—From the following list the diet of a healthy child may be arranged:

*Milk.*—This should be the basis of the diet; most children require about one quart daily. This seldom needs modification, but if somewhat difficult of digestion, it should be diluted. The milk should usually be given warm.

*Cream.*—This is of value, especially when there is a tendency to constipation. From two to four ounces of thin cream may be given daily. Above this point it should be used with caution. It should not be used upon fruits, especially sour fruits. It may be used upon cereals, upon potato, in broths, and mixed with milk.

*Eggs.*—These are a valuable form of protein. They should be fresh, soft-boiled, poached, coddled, or scrambled, but not fried. Children vary greatly as regards their ability to digest eggs; most children will take two eggs a day, some only one, and a few can not take them at all.

*Meats.*—Some form of meat should be given once a day. The best are beefsteak, mutton chop, and roast beef or lamb; next to these the white meat of chicken and certain of the more delicate kinds of fresh fish, which should be boiled or broiled. Beef and mutton should be given rare.

*Vegetables.*—Potato may be given once a day, preferably baked, with the addition of cream or beef juice rather than butter. Of the green vegetables the best are asparagus tops, spinach, stewed celery, string beans, carrots, and fresh peas. One of these vegetables should be given daily—always well cooked and mashed.

*Cereals.*—None of the ready-to-serve cereals should be given to children. They are the cause of more disturbances of digestion than almost any other common article of diet. Almost any cereal which requires

cooking may be allowed—oatmeal, wheaten grits, hominy, rice, cornmeal, farina, and arrowroot. The most important part of the preparation is thorough cooking. If the grains are used, cereals should be cooked at least three hours, after having been previously soaked for several hours. They should always be well salted, and given with milk or cream, but with little or no sugar.

*Broths and Soups.*—The meat broths are preferable to the vegetable broths. Nearly all varieties may be given. Plain broths are not very nutritious, but when thickened with arrowroot or cornstarch, and when cream or milk is added, they are very palatable, and at the same time a valuable addition to the diet. Most vegetable purées are useful, and when properly made very digestible. Beef juice may be used as directed for the second year.

*Bread and Biscuits (Crackers).*—In some form these may be given with nearly every meal, better without butter until the third year. All varieties of bread may be allowed when stale—i. e., two or three days old; also dried bread, zwieback, and oatmeal or Graham crackers.

*Desserts.*—The only ones that should be allowed up to the sixth year are junket, plain custard, rice pudding without raisins, and, not oftener than once a week, ice-cream. Of the last three, the quantity given should be very moderate.

*Fruits.*—Some fruit should be given to most healthy children every day. Oranges, baked apples, and stewed prunes are the most to be depended upon. Raw apples should not be given in most cases. Peaches, pears, and grapes (with seeds removed) may be given when thoroughly ripe and fresh, but only in moderate quantity. Much indigestion is produced by too much fruit or improper fruits. Special care should be exercised in the use of fruits in very hot weather, and in cities where they may not always be fresh. The juice of fresh berries may be given in the second year; but the whole fruit should be very sparingly given to all young children, and always without cream.

*Articles Forbidden.*—The following articles should not be allowed children under four years of age, and with few exceptions they may be withheld with advantage up to the seventh year:

*Meats.*—Ham, sausage, pork in all forms, salt fish, corned beef, dried beef, goose, duck, game, kidney, liver, meat stews and meat dressings.

*Vegetables.*—Fried vegetables of all varieties, cabbage, potatoes (except when boiled or baked), raw or fried onions, raw celery, radishes, lettuce, cucumbers, tomatoes (raw or cooked), beets (unless very small and fresh), egg-plant, and green corn.

*Bread and Cake.*—All hot bread and rolls; buckwheat and all other griddle cakes; all sweet cakes, particularly those containing dried fruits and those heavily frosted.

*Desserts.*—All nuts, candies, pies, tarts, and pastry of every description; also all salads, jellies, syrups, and preserves.

*Drinks.*—Tea, coffee, wine, beer, cider, and soda water.

*Fruits.*—All dried fruits; bananas; all fruits out of season and stale fruits, particularly in summer.

From the third to the sixth year four meals should usually be given daily and at regular intervals—e. g., 7 and 10.30 A.M.; 1.30 and 6 P.M. The second meal should, in most cases, be smaller than the others.

There are a few simple rules in feeding which should always be followed: A child should be taught to eat slowly and thoroughly masticate his food. The food must always be very finely divided, for mastication is very imperfect even up to the sixth or seventh year. It is unwise continually to urge children to eat when they are disinclined to do so at the regular hours of meals, or when the appetite is habitually poor, and under no circumstances should children be forced to eat. Indigestible articles of food should not be given to tempt the appetite when ordinary simple food is refused. Food should not be allowed between meals when it is habitually declined at meal-time. If a child refuses to eat, and examination reveals no fault with the food prepared, it should seldom be offered again until the next feeding time. In all cases of temporary indisposition, no matter of what nature, and during periods of excessive heat in summer, the amount of solid food should be reduced and more water given. If milk is the food, it should be diluted.

#### FEEDING DURING ACUTE ILLNESS.

*Infants.*—Feeding is an important part of the treatment of every acute disease in childhood, but especially so in infancy. Unless the illness is due to disease of the digestive tract, all cases must be fed in about the same way. It is much easier by proper feeding to prevent disturbances of digestion than to allay them. In infancy this complication often turns the scale against the patient. In every severe acute illness, especially if it is of a febrile character, the power of digestion is much diminished. One evidence of this is the onset with vomiting; another is the anorexia which accompanies the early stage of nearly all acute diseases. We should respect this disinclination and make it our guide in the treatment. But water is needed; withholding this will often cause the temperature to rise even higher than before.

In all acute febrile diseases the general rule should be, less food and more water than in health. For bottle-fed infants this is easily accomplished by simply increasing the dilution of the food; for nursing infants by making the nursing time shorter and giving water freely between feedings either from a spoon or bottle. During febrile condi-

tions, fat, especially, is badly borne, and this should therefore be reduced more than the other elements of the food. The diet should consist largely of carbohydrates.

Regularity in feeding is too often entirely ignored. While it is true that with some capricious children all rules must be disregarded, it is with the great majority a decided advantage to adhere to proper food and regular intervals. Food should seldom be given at less than three-hour intervals, although there is no limit to the frequency with which water may be given, and unless the stomach is irritable, almost no limit as to quantity. Stimulants, when required, are often best given in a very dilute form with the water.

*Forced Feeding—Gavage.*—Not a few cases, however, are seen in which, after a child has been several days sick, in consequence of delirium, stupor, sepsis, or some other serious condition, it may refuse all food or take so little that it is in danger of death from inanition. At this juncture forced feeding or gavage serves an excellent purpose. Both food and stimulants can thus be introduced at regular intervals with slight disturbance, and lives saved which would otherwise be lost. If gavage is employed, the stomach should first be washed. The intervals of feeding should be made at least one hour longer than is customary in health, and usually predigested foods given. Forced feeding is not applicable to chronic conditions.

*Older Children.*—The same conditions with reference to digestion exist as in the case of infants. Older patients, however, are not so easily disturbed, and the disturbance of digestion is not so likely to be serious as in the case of infants. Even here the physician should direct the food to be given at regular intervals, usually not oftener than every three hours, but should never—as is so often done—order milk to be given to the child every time he asks for a drink. In most cases, for children under five years old, milk should be somewhat diluted, usually with lime-water. Children who do not take milk readily may be given beef tea, broth, gruel, or kumyss, but rarely ice-cream or jellies so frequently prescribed, as these, if given in any considerable quantity or very often, are likely to disturb the stomach and take away what little desire for food the child may have. Raw eggs are palatable when beaten up with sherry, a little sugar, and cracked ice. Fruits, especially orange and grape juice, may be allowed in almost every febrile disease, but never given within two hours of a milk feeding.

The water given may be plain boiled water, but better, in most cases, are some of the carbonated waters, Vichy, Seltzer, or Apollinaris, these being less likely to disturb the stomach.

It is certainly a mistake to force food upon older children in any disease in which their condition is not dangerous. But when there is sepsis, delirium, or coma associated with other dangerous symptoms, gavage

may be resorted to with but little more difficulty, and with no less satisfactory results, than in infants.

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## CHAPTER V.

### *THE DERANGEMENTS OF NUTRITION.*

THE derangements of nutrition form a distinct and a very large class in the ailments of infancy, particularly during the first year. The symptoms are sufficiently definite and characteristic for them to be regarded as separate diseases, and to be discussed as such. In adults such symptoms are seldom seen except in connection with organic disease. These cases are often very puzzling, and in a large number of them a diagnosis of some constitutional disease, such as hereditary syphilis, or tuberculosis, or organic disease of the stomach or intestines, is erroneously made. At other times the symptoms resemble those of acute toxæmia. The essential condition in all these cases is the inability of the infant to get from its food what its system needs. It can not digest or assimilate enough to support life. It is unable to replace from its food the daily waste of its tissues. The constructive metabolism is imperfect; the process is, therefore, essentially one of starvation, which may be rapid or slow, according to circumstances.

The fault in these cases is partly with the organs of digestion, but principally with the food. The problem is, to adapt the food to the digestion of the individual child under consideration. The solution is often very easy at first, but the difficulties multiply rapidly the longer the condition has lasted. It is therefore essential that the true explanation of the symptoms should be recognised at the earliest possible moment. Changes occur so rapidly in very young infants that a mistake in diagnosis and a consequent delay of a few days, may be sufficient to determine a fatal result. The outcome in cases of imperfect nutrition depends almost entirely upon their management. The condition is not one which tends to right itself. Spontaneous improvement or recovery rarely takes place. In order to recognise the condition and anticipate the result, nothing is so important as a close observation of the body-weight. A child whose nutrition is a matter of difficulty should be weighed regularly, in the early months at least twice a week, and once a week throughout the first year. If this is done, the first signs of failing nutrition are unerringly detected. If an infant does not gain in weight something is wrong; a steady loss in weight is a warning which should never pass unheeded; for, unless the conditions are changed, it is practically certain to continue, and generally with increasing rapidity, until the vitality has been reduced to such a point

that no means of treatment can restore it. The younger the child, the more rapid the loss, and the longer it has continued, the greater is the danger.

For convenience of description these derangements of nutrition have been divided into three groups, differing, however, rather in degree than in kind:

1. Cases of acute inanition, which are quite rapid, generally lasting from a few days to a few weeks. They are rare except in young infants, being most frequently seen in the first three months.

2. Cases of malnutrition, in which the symptoms are much less severe than in the other groups, although they may be of long duration. While it is most common in the first two years, malnutrition may be seen at any age.

3. Cases of marasmus. This is similar to inanition, but a much slower process, lasting usually for several months. It may be seen in infants of any age.

#### ACUTE INANITION.

Inanition, or starvation, is a condition depending upon lack of assimilation. It is common in early infancy, when it often simulates serious organic disease. In older children it is not frequent, and not usually obscure. In all the acute diseases of the digestive tract many of the symptoms are due to inanition. The cases considered in the present chapter, however, are those in which there is no such association, or where the digestive symptoms, strictly speaking, are not prominent.

**Etiology.**—The essential cause of inanition is that the child does not get sufficient food, or that the food taken is not assimilated. It usually develops under one of the following conditions: (1) When a child refuses all food, whether from the breast or the bottle, or can be made to take only an insignificant amount. The cause of this it is often impossible to discover. I have seen it in a variety of circumstances, once in an infant five months old, previously healthy, who was suffering from whooping-cough. This infant utterly refused the breast, and from the spoon would take less than two ounces a day. After four days and the production of most alarming symptoms, feeding by gavage was begun, and its life, I think, saved by it. Symptoms of inanition are sometimes seen at weaning, where a child persistently refuses to take food from a bottle or spoon. (2) When the food given is entirely inadequate, as when an infant is nursing upon a dry breast, or one in which the milk supply is so scanty that the child gets practically nothing. I have occasionally seen it later, when the breast-milk, for some unexplained reason, had suddenly failed. (3) Where the character of the food is improper. On account of extreme poverty, the infant may be getting only tea, as I have known to be the case many times. Some cases



occur in young infants who are fed entirely on starchy foods. (4) Where the infant at birth has such feeble powers of digestion, because premature or delicate, that it is unable to take or to digest sufficient food to maintain life. (5) When a sudden change of food is made to one so difficult of digestion that the child is unable to assimilate it. This may happen after sudden weaning. In such cases the symptoms of inanition are mingled with those of acute indigestion, but the former usually predominate.

**Symptoms.**—The mode of development depends upon the antecedent condition. In young infants inanition often follows malnutrition where perhaps there has been nothing noticeable except a gradual loss in weight; or if the weight has not been watched, it may be observed only that the infant has not been doing well. Severe symptoms may come on quite suddenly, and if the nature and the gravity of the condition are not appreciated the case may terminate fatally in two or three days. The loss in weight is now rapid, amounting often to three or four ounces a day. The temperature in the newly born may be high, but it is more often subnormal. The pulse is always weak and rapid. The urine is scanty and very low in chlorides and often contains acetone. The extremities are cold, and the peripheral circulation poor. There is usually complete muscular relaxation, almost collapse. The skin may be dry or covered with a clammy perspiration. There is extreme pallor, and often there is cyanosis. This is always a grave symptom, and when it is marked the case usually ends fatally. Cyanosis may be present in children who have previously cried well and in whom there is no suspicion of atelectasis. The respirations are rapid and may be irregular. There may be constant worrying and fretfulness, or a condition of semi-stupor, in which the child makes no sign of wanting food. The fontanel is sunken and the pupils are contracted. The stools contain undigested food. The bowels usually move frequently, although there may be constipation, due to the small amount of food taken. When all food is refused for two or three days the stools may resemble meconium. While no desire for food is manifested, infants will sometimes swallow food when it is offered, retaining everything given for several feedings, when the whole quantity is vomited.

The course of the disease depends much upon the age of the infants. Those under one month succumb most quickly. In them the symptoms sometimes last but two or three days, seldom more than a week or ten days, the children simply drooping steadily until death occurs. With proper treatment complete recovery may take place in a week. In older infants the progress, whether upward or downward, is usually less rapid.

**Prognosis.**—The outcome of these cases is always uncertain. In few conditions is it more so. It is hard for one who is not familiar with the

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condition to appreciate the great and even the immediate danger in which a young infant may be from inanition, notwithstanding the absence of both vomiting and diarrhœa. It is difficult to estimate the gravity of an individual case except after twenty-four hours' observation. The best of all guides is perhaps the weight. Where the loss is several ounces each day the chances of recovery are small. The presence also of frequent vomiting or of diarrhœa makes the outlook very bad. A high temperature, very marked relaxation, copious perspiration, cold extremities, and cyanosis are all bad symptoms.

**Diagnosis.**—Inanition is distinguished from malnutrition by its greater severity, and from marasmus by its more acute character. The usual mistake is that of confounding inanition with some local or constitutional disease. It may be mistaken for acute indigestion, meningitis, gastro-enteritis, pneumonia, or septicæmia. The temperature when elevated is especially likely to mislead.

**Treatment.**—The existence of inanition in young infants presupposes only the feeblest powers of digestion and assimilation. If possible, a good wet-nurse should be secured, for in most of the cases the time for action is so short that there is no opportunity to experiment with artificial feeding.

The breast-milk should usually be diluted, at first with an equal volume of water or lime-water, and the quantity should be only a few drachms. It may be given with a spoon, a medicine-dropper, or a Breck feeder. If there is vomiting or diarrhœa, the milk should be pumped from the breasts, and the cream removed, since the high fat of good breast-milk is not well borne. Gradually the quantity and strength of the milk are increased until the child is allowed to take the breast in the usual manner.

When no wet-nurse can be obtained, the artificial food should be low in fat and protein and relatively high in carbohydrates. Formulas such as are desired may be obtained from whole milk. The fat and protein should be from 0.50 to 1 per cent and milk sugar, 4 or 5 per cent, and in addition maltose may be added to bring the total carbohydrate up to 7 per cent. A 5-per-cent solution of milk sugar may be given with egg albumin; or condensed milk may be tried. The quantity given should be small and the frequency not oftener than every two hours. When food is not readily taken, it may be given by gavage. Rectal feedings may be of some assistance for a short period.

Often the symptoms are due quite as much to a lack of water as to a lack of food. Injections of a normal salt solution may be given per rectum or even under the skin with very great advantage. Rectal injections should be given at 104° to 110° F. and carried high into the colon by a catheter; they should be repeated every four or five hours.

In extreme cases the slow and continuous rectal saline injection known as the Murphy method may be employed.

The other treatment required by these cases is the reduction of high temperatures by sponging or tepid baths, and the raising of subnormal temperatures by electric pads, hot-water bags, and wrapping in cotton. Stimulants are indicated, but are not very well borne; alcoholic preparations by the mouth often excite vomiting, but by the rectum they may be better tolerated. Drugs are of no use whatever.

Inanition in older infants is seldom serious unless it follows some acute illness. Peptonised milk by gavage is often useful. There are some patients, usually over a year old, who refuse fluid food of every description, and vomit it when it is coaxed or forced, yet who will take and digest in a most surprising manner some form of solid food, such as beef-steak, oatmeal, bread, crackers, or even potatoes. For the time one must give whatever the child will take, and gradually change to a suitable diet as soon as circumstances will permit. The needed water may be given per rectum.

All children who have suffered from acute inanition need the closest attention for a long time, particularly as to their feeding, regarding which suggestions will be found in the pages devoted to Infant Feeding.

#### MALNUTRITION.

Cases of malnutrition are exceedingly common, and occupy a large part of the time and attention of one engaged in practice among children. Although these children can not be said to be actually ill, they are very far from well, and their condition is often the cause of the greatest solicitude on the part of parents, not only from the existing state of health, but from the apprehension of the development of some serious organic or constitutional disease, especially tuberculosis.

**Etiology.**—Malnutrition may depend upon inherited conditions. Certain children are delicate from birth, possessing only feeble vitality, though without giving evidence of any actual disease. They are often the offspring of parents of delicate constitution and poor physical development, or of those with tuberculosis, gout, syphilis, or alcoholism. Very many city children are included in this group. Among the poor the condition is the result of bad hygiene, insufficient or improper food, overcrowding, etc. Among the well-to-do it is seen in those who inherit a too highly developed nervous organisation with a corresponding amount of physical deterioration. Another group includes those children who were premature or very small at birth, weighing perhaps only three or four pounds. Many cases are traceable to improper feeding or equally poor nursing during the first few months. These children get a poor start in life, and on that account

are handicapped throughout infancy. A frequent cause of malnutrition in infants is the pernicious custom of keeping them in close apartments where the thermometer ranges from 72° to 78° F., and where the greatest anxiety is constantly felt lest they take cold. Such infants may lose in weight, become anæmic, and exhibit all the signs of malnutrition where nothing else is wrong except the conditions mentioned. Malnutrition often depends upon some previous acute disease, especially of the stomach and intestines.

In children who are over two years old the condition of malnutrition may be due to any of the factors above mentioned—inherited feebleness of constitution, bad feeding and its resulting indigestion, too little fresh air, and close confinement indoors. It is, however, at this period much more frequently than in infancy, dependent upon some previous acute disease. As a result, an impression is left upon the child's constitution which lasts for months, often for years, and which manifests itself not by any special local symptoms, but by a general condition of debility or malnutrition. Sometimes such diseases, instead of being directly the cause of the symptoms, are the occasion which brings out some latent inherited taint or constitutional weakness in children who up to this time, perhaps, have appeared exceptionally healthy. In other cases malnutrition depends upon faulty methods in education, especially upon overpressure in schools.

**Symptoms.**—*In Infants.*—The weight is much below the average, and is either stationary or the gain is very slow, often only five or six ounces a month at a period when it should be from one to two pounds. A child under my care weighed at fourteen months but eight and a half pounds. This infant at birth weighed three and a half pounds, but in a few weeks the weight dropped to two pounds.

Not only the weight but the general physical development is much below the normal. At one year the body length may be three or four inches less than the average. Dentition is usually but not always delayed. Muscular development, too, is backward; many of these children do not sit alone until a year old, and barely walk at two and a half years. The muscles are soft and flabby, and the ligaments so weak that paralysis is often suspected. The body is so small that the head seems unnaturally large, and a diagnosis of incipient hydrocephalus is frequently made. Mentally these infants are often quite up to the average. Some symptoms of rickets may be present, but often there are none.

The examination of the blood reveals the usual changes of secondary anæmia which varies much in degree, being rarely extreme. The circulation is usually poor, the hands and feet are frequently cold. In many children the skin is unnaturally dry; in others there is a disposition to excessive perspiration, particularly about the head. Nervous symptoms are usually present. These children are restless, fretful, and irritable;

they sleep badly during the day, and often worse at night. Enlargement of the lymph glands is common, especially those of the neck. The cervical adenitis may have started from a slight catarrhal cold, but the glands continue to swell after this has subsided and may remain enlarged for months.

One of the most characteristic things about these infants is their feeble power of digestion and assimilation. Unremitting care and constant watchfulness are required to keep them up to even a moderate standard of health. The most trivial changes in food may upset them. Attacks of acute indigestion are usually brought on by overfeeding—the mistake which is almost invariably made by mothers who are discouraged with the slow progress made, and are anxious to make their children grow fat and strong. The balance is so delicately adjusted that the slightest deviation from proper rules of feeding, either as to the quality of the food or the quantity given, is immediately followed by an attack of acute indigestion, often by severe diarrhœa. As a result, the child may lose as much in two or three days as it has gained in a month or more. These acute attacks, if in summer, not infrequently prove fatal. Not only do these patients have but little resistance to acute disturbances of the stomach and intestines, but any acute disease is serious—measles, whooping-cough, and pneumonia being especially fatal.

Among the poor or in institutions, cases of malnutrition like those described, if in children under nine months old, are almost certain to go on from bad to worse until they have reached the condition described as marasmus. Between this and malnutrition no sharp distinction can be drawn; they are rather different degrees of the same general process. In private practice, where it is possible to have the best care and surroundings, with the co-operation of an intelligent mother or nurse, a very large number of these infants can be reared. After the second year has passed the problem becomes a much simpler one, and if infectious diseases and other forms of acute illness can be avoided, the probabilities are in favour of the child's becoming stronger each year and growing to maturity.

*In Older Children.*—In general appearance these children are thin, pale, and undersized, particularly if the condition is constitutional or hereditary. Sometimes they are taller than the average for their age, and their symptoms are often attributed to too rapid growth. One of the most striking things about children suffering from malnutrition is their vulnerability. They "take" everything. Catarrhal processes in the nose, pharynx, and bronchi are readily excited, and, once begun, tend to run a protracted course. There is but little resistance to any acute infectious disease which the child may contract. One illness often follows another, so that these children are frequently sick for almost an entire season. Their muscular development is poor, they tire readily,

are able to take but little exercise, and their circulation is sluggish. Mentally they are usually bright, often precocious. Many belong to the group of nervous children. They are cross, fretful, and any unusual excitement produces an effect which lasts for some time; for example, after a children's party or a Christmas tree they may lie awake half the succeeding night, and may be really ill for two or three days. Their sleep is usually disturbed and restless; they waken frequently, and occasionally suffer from night-terrors. At a later age they are favourable subjects for chorea, neuralgia, and all functional nervous disorders.

Digestive symptoms, if not constant, are very easily excited. In fact, they do not suffer so much from chronic indigestion as from a delicate or feeble digestion, which is easily upset by the slightest deviation from the regular routine. Children of five or six years have to be fed as carefully as infants of eighteen months or two years. The appetite is usually poor, and mothers are distressed because their children eat so little, yet, when food is urged upon them, attacks of indigestion follow with singular uniformity. The tongue is slightly coated the greater part of the time. The bowels are apt to be constipated, apparently more from lack of muscular tone than from anything else. From time to time, from slight causes, such as exposure to cold, or even from fatigue, there may be large quantities of mucus in the stools for two or three days at a time, although this is not a prominent feature of most of these cases. When they are not fed with the greatest care these children suffer constantly from indigestion. A moderate amount of anæmia is always present, and this may be the most striking feature.

The duration of the condition depends very much upon the cause. If the cause is constitutional or inherited, it is likely to last throughout childhood, but it often greatly improves about the time of puberty. Where it follows some acute illness it commonly lasts for a few months only; but the effect of an acute attack of broncho-pneumonia or of ileo-colitis may continue for years. If the malnutrition is the result only of the child's surroundings, like the confinement incident to city life, very rapid improvement may follow a removal to the country. In some children marked improvement is seen about the seventh year; in others, a great change comes at puberty.

**Diagnosis.**—The physician should not be too ready to make a diagnosis of simple malnutrition. Before accepting such a diagnosis, he should examine the child with the greatest care, to exclude the common organic and constitutional diseases. Much regarding inherited constitutional tendencies can be learned from the family history and from the condition of other children in the family. In the first place, tuberculosis must be excluded by a study of the temperature and physical signs rather than by the tuberculin test. This often gives a positive reaction when no other evidence of this disease exists and when none develops

subsequently. It is in such cases extremely doubtful whether the latent tuberculous focus plays any part in the production of the symptoms. Other things to be considered are syphilis, rickets, chronic malarial poisoning, diseases of the blood, intestinal parasites and of course organic diseases of the lungs, heart, stomach, intestines, liver, and kidneys. Even malignant disease, though rare, should not be overlooked. It may take careful observation for several days, and sometimes for weeks, with repeated physical examinations, before all these conditions can be positively excluded.

The next step in the diagnosis is to discover upon which one of the many possible causes malnutrition depends. In private practice the great proportion of cases are due to improper feeding or nursing; next in importance are improper surroundings; and last come inherited constitutional conditions. In other words, most of these children are born healthy, but become ill or delicate in consequence of improper management.

In older children, after excluding constitutional and local diseases, the whole life of the child must be investigated to discover the fundamental condition which is at fault. A carefully obtained history from infancy is of the greatest assistance. It is often difficult, and sometimes impossible, to get at the primary factor, for in cases of long standing there may be symptoms connected with almost every function of the body. One should scrutinise closely the quality and quantity of food given, the amount of sleep, the hours of study and recreation, the amount of exercise in the open air, and the physical conditions surrounding the child. Usually the most important factor in the case can be discovered.

**Prognosis.**—This depends much upon the cause of the condition; if it is one that can be removed, the prognosis is good not only for improvement but for complete recovery. The longer the condition has lasted and the greater the general disturbance the slower will be the improvement. The great danger is the supervention of some acute disease while the child's resistance is so greatly reduced. Acute indigestion, gastro-enteritis, and broncho-pneumonia are especially to be dreaded.

Since everything depends upon the fidelity with which directions as to diet and general management are carried out, the cases which present the greatest difficulties are those in which these conditions are hardest to control. When a child is not only suffering from malnutrition, but has been indulged and spoiled in every way by anxious but unwise parents, no success is to be expected unless the child can be placed in the hands of an experienced and trustworthy nurse. Cases due to improper feeding or to bad surroundings usually improve when these are corrected, and the worse these conditions have previously been the greater the improvement to be expected. Those depending upon an inherited,

delicate constitution are not so hopeful, and require the closest attention throughout childhood.

**Treatment.**—This is a problem of nutrition to be solved by diet and general management, drugs occupying a very small place.

*In Infants.*—In very young infants treatment is chiefly a question of feeding. This should be carried on according to the rules given in the chapter upon Feeding in Difficult Cases. These children often do fairly well during the first year, but after this time frequently do very badly, on account of the failure to appreciate the fact that, although over twelve months old, in point of development they resemble healthy infants of four or five months, and are to be managed as such. If they are nursing, weaning should often be deferred until the sixteenth or eighteenth month, or at least partial nursing should be continued until that time. When cow's milk is begun it should always be very largely diluted, usually modified as for a healthy infant a few months old. Very rarely a child is met with who has an idiosyncrasy as regards cow's milk and can not take even the smallest amount without marked disturbance. I have seen a single feeding in which one ounce of milk was given, and that diluted three times, produce a violent attack of acute indigestion which proved well-nigh fatal. Feeding during the entire second year should be carried on very much as in ordinary healthy children from the sixth to the twelfth month. A deviation from this rule almost invariably results disastrously. One must be guided as to the amount and character of the food, not so much by the child's age as by his digestive capacity, and in most cases this is much feeble than the mother or even the physician supposes. In many of these cases, cow's milk—for them the most valuable of all foods—has been excluded from the diet, when the only trouble is that it has not been given in sufficient dilution. For some children it may be partially peptonised during periods when digestion is especially feeble.

Next in importance to diet is fresh air. Often these patients will not improve with any variation in diet until fresh air is secured. Then increased digestive power is seen in the course of a few weeks, sometimes in a few days. The natural tendency of a mother who has a delicate infant, or one suffering from malnutrition, is to house it closely and never allow it a breath of fresh air. It is of the greatest assistance if these children can be sent to a warm climate for the winter. If this is not possible, fresh air may be obtained by changing apartments, or by an airing in the room with the windows open. In the beginning this should be done for a few minutes only, the time being gradually increased to two or three hours each day. The child should be clothed as for the street, and, if necessary, hot bottles should be placed at the feet.

Cold sponging is another valuable tonic. After the morning bath is



given, at 95° F., the entire body should be sponged for a moment only, with water at a temperature of 60°, or even 55° F. This produces a certain amount of shock and causes loud crying, which is of itself beneficial. How frequently this should be done will depend upon the reaction following it. If the child remains blue and cold for some time afterward, the cold sponging should not be repeated. If there is a good reaction, it may be used daily.

Friction and massage are useful in many cases. The child should be laid upon the lap of the nurse, if possible before an open fire, and should always be covered with a blanket. The entire body should then be rubbed for ten or twenty minutes with the bare hand, or, better, with cocoa butter. Simple rubbing may be used, or the movements of massage employed. If the latter, they should be very gentle at first, and only for a short time. Professional operators are inclined to be too energetic for little children. There is no advantage in rubbing with cod-liver oil instead of cocoa butter, while the odour makes it decidedly objectionable.

The only tonics I have found of much value are iron, nux vomica, and cod-liver oil. Nux vomica may be given alone or with wine. Cod-liver oil is too much used in these cases, and in too large doses. Many of these infants can not take it at all. It should rarely be given when the tongue is coated and the appetite very poor. The dose should always be small, e. g., ten drops of the pure oil three times a day, or twice as much of an emulsion. Olive oil in many cases is better borne and quite as efficacious; it may be given in half or teaspoonful doses three times a day.

The secret of success in treating cases of malnutrition is, to hold the patient to a regular routine in feeding, sleeping, and in everything relating to his life. Experiments are nearly always unfortunate. The physician should lay down in writing, for the guidance of the mother, specific rules with regard to the amount of food, the time at which it is to be given, the hours for bathing, sleep, and airing. He should see the patient at regular intervals and often enough to be sure that his orders are being enforced. Good results are obtained only by constant watchfulness, and although improvement may not be seen at once, it is in most cases sure to come if the mother will co-operate. In my own experience no class of patients have given me so much satisfaction as cases of malnutrition in infancy.

*In Older Children.*—The same general principles are to be applied to them as to infants. The diet is of the first importance. Only the simplest, plainest, and most easily digested articles of food should be given. The problem is to secure the maximum nutritive value in the food with the minimum tax on the digestive organs. Milk, beef, eggs, the lighter and more easily digested cereals, bread, and fruit should form the diet. All sweets, pastry, highly seasoned food, candy, nuts, tea,

and coffee should be absolutely prohibited, and, in fact, all the articles mentioned as "forbidden" in the chapter on the Feeding of Older Children. When the appetite is poor and simple food not well taken, the child should not be allowed to take indigestible articles for the sake of eating something. Nothing should be given between meals, and regular hours of feeding must be followed. Usually I have found three meals a day, for children over three years old, better than the practice of giving more frequent feedings. But this is not always the case. Under no circumstances should children be coaxed, urged, or hired to eat; much less should they be forced to do so. There is a popular misapprehension in regard to the variety in diet which children need. Most cases do better when a very simple and fairly uniform diet is continued.

The nervous factor is a very large and a very important one. Many of these children are essentially cases of neurasthenia at as early an age as four or five years. Excitement and activity are what they crave and what must be most carefully avoided.

The general habits of children should be directed; there should be regular and early hours for retiring, freedom from undue excitement, and interest should be awakened in out-of-door amusements. A pony or dog will be found useful. Children should be kept as much as possible in the open air, but the amount of active exercise should be strictly limited. Usually they do much better if they can be in the country during the entire year. Only a limited amount of reading and study should be allowed; and if children are at school, care should be taken that overpressure is not the cause of the symptoms, particularly in an ambitious child. The cold sponging given in the morning, as described in the introductory chapter on General Therapeutics, is extremely beneficial to children who take cold readily. Massage is useful for the benefit which it affords to the chronic constipation which is so frequently a symptom of malnutrition.

Of the tonics, iron, arsenic, and cod-liver oil are required in most cases, and the amount and combination may be varied from time to time, with the season of the year and the condition of the child's digestion. In general, these children require early hours, a simple diet, a quiet, regular life, and very little medicine.

#### MARASMUS.

*(Infantile Atrophy; Simple Wasting.)*

Wasting is a symptom of many conditions in infancy. It occurs in tuberculosis, in infantile syphilis, and also as a result of acute or chronic disease of the stomach and intestines. Cases of wasting dependent upon such causes are not included in this chapter.

Marasmus is the extreme form of malnutrition seen in infancy, oc-

curring, so far as is known, without constitutional or local organic disease. It is a vice of nutrition only.

**Etiology.**—Marasmus is not very often seen in the country or in private practice; but it is frequent in dispensary practice in all large cities, and is especially common in institutions for young infants. In my own experience in four institutions, more than one half the deaths under one year were directly or indirectly from this cause. Marasmus is a very large factor in the immense infant mortality of large cities in summer. Although the cause of death is usually reported under some other name, the determining factor in the fatal result is the previous marantic condition of the patient. The primary cause may be a congenital weakness of constitution which may depend upon heredity. It is often seen in premature children and in the illegitimate offspring of girls of sixteen or eighteen. In the vast majority of cases, however, it depends upon two factors—the food and the surroundings. Among the poor who live in tenements, infants who are artificially fed almost invariably do badly. This is due to ignorance in regard to the proper methods of infant feeding and inability to procure what the child requires, especially pure cow's milk. A country infant may be neglected in many respects, and is often badly fed; but he has plenty of pure air, and usually thrives. In the city, as long as an infant has a plentiful supply of good breast-milk he continues to do well in most instances, in spite of the fact that his surroundings are bad. When there are not only bad feeding and unhealthful surroundings, but also an inherited constitutional vice, we have all the factors required to produce marasmus in its most marked form. The odds are so against the infant that the feeble spark of vitality flickers for a few months only and gradually goes out.

Another prominent factor in the production of marasmus is the overcrowding of infants in institutions. Even though artificially fed after the most approved methods, I have seen scores of infants who were plump and healthy on admission lose little by little, until at the end of three or four months they had become wasted to skeletons—hopeless cases of marasmus, dying of some mild acute illness, such as an attack of indigestion or bronchitis, the essential cause, however, being marasmus. The common mistake is that of placing too many children in one ward, with no chance of obtaining a proper amount of fresh air. No house-plant is more delicate or sensitive to its surroundings than is an infant during the first few months of life.

**Lesions.**—The post-mortem findings in cases of marasmus are exceedingly unsatisfactory, and throw little if any light upon the disease. Every now and then general tuberculosis is discovered in patients dying apparently of marasmus, the existence of which was not previously suspected. An occasional lesion is fatty liver. This may lead to such

enlargement of the organ that its weight is increased by one half. Both to the naked eye and under the microscope the usual changes of fatty infiltration are present, often to an extreme degree. In the past too much has doubtless been made of this condition of the liver in marasmus. From figures given elsewhere (see article on Fatty Liver), it will be observed that the lesion is not more frequent in this condition than in infants dying from other diseases. The most marked examples are seen in cases of marasmus which have lasted for seven or eight months. Its exact relation to the condition of wasting has not yet been determined.

With these exceptions the autopsies show nothing striking, and I have had the opportunity to make at least two hundred of them. The lesions usually found are the following: The brain is commonly anæmic, with dark fluid blood in the sinuses, marantic thrombi being rare. A strip of hypostatic pneumonia, from one to two inches wide, may be seen along the posterior border of both lungs, involving the lung to the depth of half an inch, or less. In the younger infants there are frequently areas of atelectasis in the lower lobes. The pleura is almost invariably normal. The heart is pale, with perhaps a slight increase in the pericardial fluid. The spleen and kidneys are pale, but otherwise normal. The stomach may be dilated; the mucous membrane is usually pale, often coated with tenacious mucus. The intestines contain undigested food, sometimes mucus. The solitary follicles of the colon and small intestine, and sometimes Peyer's patches, are slightly enlarged, the mucous membrane in other respects being normal. The mesenteric glands are often slightly enlarged. In addition to the above, there may be evidence of some recent infection, which has been the cause of death; there may be acute bronchitis, broncho-pneumonia, or intestinal catarrh.

The above lesions represent what has been found in the great majority of the cases, and very disappointing they are to one who sees them for the first time. Nor does the microscopical examination of the organs throw any light upon these cases. I have personally examined with care the stomach and intestines of more than a dozen cases, several of them in which autopsies were made only two or three hours after death, without finding anything of pathological importance. The theory advanced, that atrophy of the intestinal tubules is the explanation of marasmus, has found little support.

The condition of marasmus seems rather to be a failure of assimilation, owing to imperfect digestion, improper food, unhygienic surroundings, or feeble constitution. As a result, there is a progressive loss in weight, feeble circulation, imperfect lung expansion, imperfect oxidation of the blood, lowered body temperature, and, finally, a deterioration of the blood itself. Each of these effects becomes in turn a cause aggravating all the others, continuing until a condition is reached which

is incompatible with life, for resistance becomes so feeble that the slightest functional disturbance proves fatal.

**Symptoms.**—The general history of these cases is strikingly uniform. The following is the story most frequently told at the hospital: "At birth the baby was plump and well nourished, and continued to thrive for a month or six weeks while the mother was nursing it; at the end of that period, circumstances made weaning necessary. From that time



FIG. 32.—MARASMUS; A PATIENT IN THE BABIES' HOSPITAL, TEN MONTHS OLD, WEIGHT SIX POUNDS. Weight at birth reported to have been nine pounds.

the child ceased to thrive. It began to lose weight and strength, at first slowly, then rapidly, in spite of the fact that every known form of infant-food was tried." As a last resort the child, wasted to a skeleton, is brought to the hospital.

The most constant symptom is a steady loss in weight until a condition of extreme wasting is reached, at which point they may remain for many weeks. The general appearance of these patients is character-

istic. They have an old look; the skin is wrinkled, has lost its tone, and hangs in folds upon the extremities (Fig. 32). The legs are like drumsticks; the abdomen is prominent; the temples are hollow; the fontanel is sunken; the eyes large; the features sharp; and the hands resemble bird-claws. Often the children are reduced literally to skin and bones. Anæmia is a very marked and almost a constant symptom, the amount of hæmoglobin being frequently reduced to thirty per cent, and in one of my cases to eighteen per cent. Anæmic heart-murmurs are frequently heard. The body temperature is usually subnormal, unless artificial heat is used. A rectal temperature of 95° or 96° F. is very common, and one of 93° or 94° F. is occasionally seen. In addition to the pallor of the face, there may be a leaden hue due to congenital or acquired atelectasis. A frequent symptom is general œdema. The first thing which calls attention to this is often an unexpected gain in weight. The œdema may increase until the cellular tissue of the whole body is affected. I have never, however, seen effusions into the large cavities. Œdema is usually associated with marked anæmia, and is generally a bad symptom. The stools are sometimes normal, but usually contain undigested food, and are large in proportion to the amount of food taken. No matter how carefully fed, these patients are easily upset. Now and then mucus is seen in the stools, but this is neither a constant nor a marked feature. Vomiting is excited from the slightest cause, and often food is regurgitated almost as soon as swallowed. The appetite, in a severe case, is almost entirely lost; children refuse to take food from the bottle or spoon, and unless fed by gavage they die of inanition. In the earlier cases there may be an unnatural hunger, so that the children cry much of the time, and are relieved only when the bottle is given.

The complications are thrush, erythema of the buttocks, and bedsores, sometimes over the sacrum and heels, but most frequently upon the occiput. Occasionally there is seen a reflex spasm of the muscles of the neck, producing a marked opisthotonus, which may last for several days or weeks.

The course of the disease in most cases is steadily downward. It may be cut short at any time by acute disease. Frequently these infants die suddenly when apparently they are as well as they have been for several weeks. In many instances the autopsy reveals no explanation of the sudden death; but in other cases it may be due to the regurgitation of food, and its aspiration into the larynx, the patient being too weak to cough. Rarely, death occurs from convulsions. In summer, these children wilt with the first days of very hot weather, and die often in a few hours from a slight functional derangement of the stomach and bowels.

**Diagnosis.**—No sharp line can be drawn between marasmus and malnutrition. In the wasting which follows chronic disease of the stomach and intestines there is usually a history of an antecedent acute attack.

Not infrequently tuberculosis is found at autopsy, even in infants of a few months, in whom there have been no symptoms except those of marasmus; but during life tuberculosis may now be recognised in most cases by the von Pirquet skin test.

**Prognosis.**—This depends on the age of the infant and the extent and duration of the disease. If the child is over eight months old, the chances of recovery are much better than in one under four months, for the fact that it has lived so long is generally evidence of pretty strong vitality. Very young infants are always difficult subjects to deal with. They go down more rapidly, and build up more slowly than those who are older. In most other circumstances the prognosis is much worse in cases of long duration. In a given case much depends upon whether everything possible can be done for the child: whether a wet-nurse can be secured or artificial feeding done in the best manner, and whether the patient can have the benefit of the best surroundings, in the country in summer and in winter a warm climate where it can be kept out of doors the greater part of the time. In institutions cases under four months old are usually hopeless. Of those over eight months quite a proportion can be saved by proper treatment, even though the body-weight is reduced to eight or nine pounds. When recovery occurs it may be complete, and the child at two or three years may be as vigorous as any child of its age. All these statements refer only to cases of simple marasmus. The presence of organic disease puts the case into another category.

**Treatment.**—The most important is that which relates to prophylaxis. Maternal nursing should be encouraged by every possible means especially among the poor. For those who must be artificially fed the important things are a pure milk supply together with proper instruction as to how it is to be used in infant feeding. At the same time opportunities for fresh air should be secured. This is a large part of the difficulty in institutions. At least one thousand cubic feet per patient should be secured with proper ventilation and, what is almost as essential, adequate nursing.

As far as possible, wet-nurses should be obtained if the infants are under four months old. For these very young patients success by artificial feeding is generally impossible. With those of six months or over, good artificial feeding is very frequently successful. In modifying cow's milk for these cases the formulas most likely to agree are those with low fat, low protein—partially peptonised in many cases—and relatively high sugar. Further suggestions will be found in the chapter on Feeding in Difficult Cases. In institutions we seldom succeed without wet-nurses.

For very young infants, with a temperature which is habitually sub-normal, some means of maintaining the body heat must be employed. The simplest and usually an effective means is to oil the body and en-

velop it completely in a cotton jacket and then surround it with hot-water bags or bottles. The general management should be much the same as described in the chapter on Malnutrition. They require no drugs, but a great deal of careful nursing.

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## CHAPTER VI.

### *DISEASES DUE TO FAULTY NUTRITION.*

THE diseases due to faulty nutrition are numerous. There are two, however, which have been so clearly shown to originate in this way that they may be put in a class by themselves. These are scorbutus and rickets. The prevailing opinion of the medical profession is that both of these are essentially "food-diseases." The purpose of considering them in connection with the disturbances of nutrition is to emphasise this relationship.

#### SCORBUTUS (*Scurvy*).

Scorbutus is a constitutional disease due to some prolonged error in diet. It is characterised by spongy, bleeding gums, swellings and ecchymoses about the joints, especially the knee and ankle, hæmorrhages from the nose, and occasionally from other mucous membranes, extreme hyperæsthesia, and often pseudo-paralysis of the lower extremities. Added to these local symptoms there is in advanced cases a general cachexia with marked anæmia. While scorbutus and rickets are very frequently associated, they can not be considered as different forms of the same disease. Cases of scorbutus were, however, described in older writings under the title of Acute Rickets.

Scurvy was well recognised and graphically described by Glisson as long ago as the middle of the seventeenth century. For our modern knowledge of the pathology of this disease we are indebted to the observations of Barlow and Cheadle. On the continent of Europe scurvy is most frequently known as Barlow's disease.

For the statistical matter here presented I am indebted to the report of the American Pædiatric Society's Collective Investigation of Infantile Scurvy in 1898, embracing 379 cases, reported by 138 observers. Of these, 31 cases were from my own practice.

**Etiology.**—Age is an important factor; more than four-fifths of the cases occur between the sixth and the fifteenth months, and half of them between the seventh and the tenth months. Scurvy has been seen in infants under a month old. The great majority of the cases reported have been observed in private practice, often in the best surroundings. Previous disease is not a factor of much importance. Most of the chil-



dren attacked have been in good health up to the development of scurvy. In about one-fourth of the number some previous derangement of the digestive tract has existed.

The only etiological factor yet known to bear any constant relation to the production of scurvy is diet. The important facts regarding the previous diet brought out by the Society's investigation are as follows:

|               |   |                            |                           |
|---------------|---|----------------------------|---------------------------|
| Previous food | { | Breast-milk . . . . .      | in 12 cases; alone in 10. |
|               |   | Raw cow's milk . . . . .   | " 5 " " " 4.              |
|               |   | Pasteurised milk . . . . . | " 20 " " " 16.            |
|               |   | Condensed milk . . . . .   | " 60 " " " 32.            |
|               |   | Sterilised milk . . . . .  | " 107 " " " 68.           |
|               |   | Proprietary infant-foods   | " 214 "                   |

This table shows that while scurvy may occasionally develop with almost any variety of food, three stand out prominently—viz., proprietary infant-foods, condensed milk, and sterilised milk. In all of these it would appear that something needed for normal healthy nutrition is wanting. Scurvy is not likely to follow unless an improper diet is continued for a long period, usually several months. In some instances where it developed in nursing infants, the nurse's milk has been examined and found totally inadequate to the needs of nutrition, many of the children having exhibited serious disturbances of nutrition before any signs of scurvy appeared.

In several of the cases reported as occurring with a diet of raw or pasteurised milk it seems certain that the milk formula used was at fault, the most common error in those I have seen being low protein. Several cases have come under my personal observation where children had been kept for four or five months upon percentages which should have been continued only a few weeks. However, I have seen at least three cases of scurvy which developed while taking pasteurised milk where the percentages employed could hardly have been the explanation, and the heating (167° F. for thirty minutes) seemed to be the cause. However, I believe scurvy to be an exceedingly rare result of the pasteurisation of milk, so rare, indeed, as not to be weighed against its immense advantages. With the lower temperature now generally employed (155° F.) it need not be feared. The number of cases occurring while upon a diet of sterilised milk (usually heated to 212° F. for one hour) is so large that we are driven to the conclusion that the heating alone was the cause, especially since prompt recovery has frequently followed when no other change was made than to discontinue the heating. These facts show that sterilised milk should not be continued as the sole diet for long periods—i. e., for several months—and that its possible danger should be kept in mind.

No one fact in the etiology of scurvy is better established than its development after the prolonged use of condensed milk or the propri-

etary infant-foods. Scurvy occurs not only when the foods are used with condensed or with sterilised milk, but also, though less frequently, with fresh milk. The inference is that these preparations cause scurvy not only by what they lack, but possibly by something which they contain. Some have ascribed the results to the ferments present. This view has some support in the occurrence of scurvy after the prolonged use of peptonised milk, an infrequent but a well-established fact. In this respect, as with reference to sterilised milk, my personal experience, including now nearly one hundred cases of scurvy, coincides with the findings of the Society's report.

While it may be regarded as established that the cause of scurvy is dietetic, no single dietetic error can be held responsible for the disease. None of the theories yet advanced in explanation of how diet causes scurvy is wholly satisfactory.

**Lesions.**—The most marked effects of scurvy are seen in the bones, blood-vessels, and the blood. The number of recorded autopsies is not large, only six being included in the Society's report. I have myself had the opportunity of making examinations in three cases. The findings are remarkably uniform, but represent, of course, the extreme results of the disease. The most striking lesion is subperiosteal hæmorrhage, which is practically constant and may occur almost anywhere in the body, but affects chiefly the bones of the lower extremities; it is often very extensive, and may reach from the knee to the great trochanter, or from the ankle nearly to the knee. Extravasations may also be found between the muscles, and blood may infiltrate the cellular tissue in the neighbourhood of the joints. Besides these lesions resulting from hæmorrhagic periostitis the bone itself may be affected. Separation of the epiphysis from the shaft of some of the long bones, generally at the lower end of the femur or lower end of the tibia, is found in most of the fatal cases. Notwithstanding the serious lesions near the large joints, the joints themselves are usually normal.

The minute bone changes are somewhat similar to those of rickets. But there are also differences of importance. The disposition to hæmorrhage, which is altogether the most characteristic feature of scurvy, is entirely wanting in rickets. The visceral lesions are inconstant. Those most frequently found are small hæmorrhages beneath the pleura, pericardium, and peritonæum, sometimes into the various organs, also broncho-pneumonia, and nephritis. There may be small extravasations found upon the surface of any of the mucous membranes. The alterations in the blood-vessels are undoubtedly an important factor in bringing about the disposition to hæmorrhage, but as yet they have been very imperfectly studied. The changes in the blood, in the gums, and the lesions of the skin will be considered with the symptoms.

**Symptoms.**—In many cases a period of indisposition, fretfulness, pallor, and failing nutrition precedes the local symptoms, but usually tenderness of the legs is the first symptom noticed. In the beginning this is occasional and so slight as to cause the infant to cry only upon being handled. Later it becomes almost constant and is very acute. At first this soreness is not very definitely localised, but is generally more marked about the knees and ankles. Some swelling may be noticed, often just above the ankle-joints. Coincident with these may be seen the changes in the mouth. The gums are of a deep purplish colour, swollen, particularly about the upper central incisors, and may quite cover the teeth. They bleed from the slightest irritation, and sometimes spontaneously. The child now becomes fretful and cross, sleeps badly, loses colour, weight, and appetite. He may become quite cachectic in appearance. All these symptoms come on very gradually, often with periods of a few days in which apparent improvement is seen. Sometimes they may continue for several weeks without making any perceptible impression upon the child's previously good condition.



FIG. 33.—SCURVY SHOWING CHARACTERISTIC SWELLINGS AND POSTURE. Patient 8½ months old, fed exclusively upon malted milk after the age of 3 months. Epiphyseal separation at the upper extremity of both humeri, lower extremity of both femora and lower extremity of left tibia. Prompt and complete recovery.

If the disease is recognised, and proper treatment instituted, rapid improvement follows, with complete and permanent recovery. If not recognised, and the faulty diet is continued, the disease advances to the more severe form. The tenderness of the legs becomes exquisite, so that any movement or even the slightest touch causes the child to scream with pain or apprehension. The posture is very characteristic. There is semiflexion of thighs and legs and outward rotation at the hip. (See Fig. 33.) In this position the child often lies motionless and voluntary movements of the extremities can not be excited. Paralysis is often suspected. The disability is chiefly owing to the extreme pain which motion provokes, but may depend upon epiphyseal separation. Small ecchymoses are frequently seen about any of the large joints, resembling

the ordinary "black-and-blue" spots, and these often confirm the opinion previously formed that the child has met with some accident. The swelling near the joints, particularly the knee, may be so great that the limb is nearly twice the size of its fellow. The mouth symptoms are usually striking. In addition to spongy, swollen, bleeding gums, dark purplish bags may be seen over teeth not yet through. There may be bleeding from the roof of the mouth or from the pharynx. The pain is sometimes so severe as seriously to interfere with taking food; there is moderate though rarely extreme salivation. Blood may be vomited or passed with the fæces or the urine. In the severe cases the stools are rarely normal, more or less catarrhal colitis usually being present. The general condition is one of grave anæmia, accompanied by a marked cachexia and progressive wasting. The child cries almost constantly, sleeps little, and is truly a pitiable object. Slight fever is often present. Unless recognised and the cause removed, the condition grows steadily worse, the symptoms continuing until death occurs either by a slow asthenia, or suddenly from heart failure, or from some intercurrent disease, such as broncho-pneumonia or acute gastro-enteritis. The duration of the illness in the fatal cases is from two to four months.

The onset is gradual in the great majority of the cases, the earliest symptoms noticed in the order of frequency being pain and tenderness of the legs, soreness and sponginess of the gums, disability, anæmia, cutaneous hæmorrhages, and very rarely hæmaturia.

Pain and tenderness are very prominent, being noted in 95 per cent of the Society's cases; in the majority they were present only on motion or handling. The location of the pain and tenderness in 184 cases was as follows: Lower extremities alone, 133; upper extremities alone, 2; lower and upper, 42; lower and trunk, 7. In all but two cases, therefore, the lower extremities were affected, the lower part of the thigh and the leg just above the ankle being the usual seat.

Disability, or pseudo-paralysis, is a very common symptom, and in all severe cases a constant one. It exists in varying degrees from a slight disinclination to use the limb to complete helplessness. In many cases it is more marked than the pain, and has led to a diagnosis of poliomyelitis.

Swellings are associated with pain and tenderness in most of the severe cases. They are most marked near the joints, but may extend for some distance along the shafts of the bones. In nearly all cases the location is the lower part of the thigh or the lower part of the leg, and usually of both sides. Swellings are occasionally seen near the wrists, elbows, shoulders, and hip-joints; in rare cases, over the ribs, scapula, or ilium. Redness is not generally present, but the parts may have a dark purplish colour. It is to the hæmorrhages that both the swellings and the discoloration are chiefly due.

Protrusion of the eyeball is present in about ten per cent of the cases; an extreme exophthalmus is sometimes seen, and is due to orbital hæmorrhage.

The gums are affected in nearly all cases, the exceptions being those recognised and treated early. Hæmorrhage occurs in about one-half the cases, and frequently there is ulceration not unlike that of a mercurial stomatitis. It is rather curious that, though the lower teeth are cut first, the upper gum is almost always most affected, and in the milder cases usually alone involved. Of 45 cases in which no teeth had been cut, the gums were affected in 24 and normal in 21. This is sufficient to disprove the old opinion that the gums are affected only when teeth have appeared. The severe inflammation and ulceration sometimes seen seem to be the result of secondary infection.

Hæmorrhages beneath the skin are present in about half the cases. They are rarely extensive, usually multiple, and their location is no doubt often determined by a slight traumatism. Hæmorrhages from the mucous membranes are not quite so frequent. There may be bleeding from the gums, nose, bowels, kidneys, and rarely from the stomach. Hæmorrhages in most cases are frequently repeated, but seldom profuse.

Epiphyseal separation is seen only in very severe cases. It is most frequently either of the lower epiphysis of the femur or the tibia, or the upper epiphysis of the humerus, and is often bilateral. The actual separation may be caused by some slight injury, the condition of the bone predisposing to this occurrence. In three cases of my own with separation which recovered, rapid union occurred under anti-scorbutic treatment.

Anæmia is slight in the early stage, but increases as the disease progresses. Blood examinations may show great reduction of the hæmoglobin, sometimes to thirty-five or forty per cent; also in nearly all cases a proportionate reduction of the red cells. The changes are those of an ordinary secondary anæmia.

The urine contains albumin in one-fourth of the cases; in nearly half of those containing albumin casts also are found. In rare cases hæmaturia has been an early symptom. Blood cells usually in moderate numbers are found in practically all but the mildest cases, and are of some diagnostic importance.

Evidences of general malnutrition are present in all advanced cases, varying, of course, greatly in degree. In a few infants under my own observation the weight, colour, and general appearance of health have continued in spite of very decided local symptoms. In most of them the impaired nutrition is shown by loss of appetite, occasional attacks of vomiting, and still more frequently by derangements of the bowels, which vary from slight indigestion to a serious catarrhal condition of

both small and large intestine. It is with the latter that the discharge of blood is usually seen.

**Association with Rickets.**—In the Society's investigation great pains were taken to obtain definite and accurate data regarding this. Of the cases, 340 in number, in which this point was noted, symptoms of rickets were present in 152, or 45 per cent; these symptoms were recorded as slight in 72; marked in 64; and not specified in 16. In the remainder of the cases, 55 per cent, it is definitely stated that symptoms of rickets were absent. It is also stated that in 50 of the patients which were rachitic, the rickets antedated the development of the scurvy. From these facts it would seem to be pretty well established that though rickets and scurvy have points of resemblance, such as the age when they are seen, bony changes, dependence on defective nutrition, etc., they can not be regarded as different forms of the same disease. The two most striking characteristics of scurvy—viz., tendency to hæmorrhages and prompt curability by fresh food and fruit juices—have no counterpart in rickets. However, their co-existence in the same patient is of common occurrence.

**Diagnosis.**—The disease with which infantile scurvy is most frequently confounded is rheumatism. In fully four-fifths of the cases which have come to my own notice this has been the previous diagnosis. The extreme rarity of rheumatism under one year should always make one cautious; pain and tenderness of the legs only, should, in an infant, invariably suggest scurvy rather than rheumatism. The extreme disability has often led to a diagnosis of poliomyelitis, but here again the acute tenderness should set one right. Many cases of scurvy come into the hands of the orthopædic surgeon with a diagnosis of joint or spinal disease. Where the swelling was mainly of one limb I have twice known a diagnosis of malignant disease to be made, from the cachexia, the shape of the swelling, the discoloration, and the pain. I have known two cases to be operated upon by eminent surgeons, once with a diagnosis of sarcoma and once of osteitis of both tibiæ. Not until the subperiosteal hæmorrhages and epiphyseal separation were discovered was the nature of the trouble suspected.

The diagnosis of scurvy seldom presents any difficulties to one who has once seen a case. No one need err if the essential features of the disease are kept in mind: the extreme soreness of the legs, spongy, swollen gums, swelling near the large joints, a tendency to hæmorrhages, and usually a history of the prolonged use of some proprietary infant food, or sterilised or condensed milk. The epiphysitis of hereditary syphilis has many symptoms in common with scurvy, but it usually occurs at an earlier age (before the fifth month) and other evidences of syphilis are usually present. If any doubt exists, this will be removed by the prompt improvement and generally rapid cure following an anti-scorbutic diet.

**Prognosis.**—This is invariably good if the disease is recognised early. No patients with symptoms so serious improve with such marvellous rapidity as do the great majority of those with scurvy, under proper management. The figures of the Society's report on this point are interesting. The average duration of the disease before treatment was begun in over three hundred cases was somewhat over three weeks. In eighty per cent striking improvement was noticed during the first week of treatment, and in forty per cent within three days. Over two-thirds of these cases were well within three weeks, and nearly one-third within one week, after the beginning of treatment.

It is only when the disease is of long standing, when the malnutrition is severe, or when serious complications, usually involving the digestive tract, are present that the symptoms persist and the issue becomes doubtful. It is difficult to tell what the exact mortality of scurvy is. Any case allowed to go on may result fatally. The younger the infant the more likely is this to occur. I have seen four deaths in nearly one hundred cases. In one of my patients death resulted from hæmorrhage which followed an incision into an epiphyseal swelling at the lower end of the femur, made before I saw the patient, and which persisted despite all treatment. Barlow's early article included thirty-one cases with seven deaths. It is rare that scurvy leaves any permanent effects. Recovery is not only rapid but complete. Relapses are extremely rare and have been observed only in one or two cases, where chronic indigestion existed of so extreme a character that proper feeding was impossible. The after-effects are usually the result of prolonged malnutrition, of which the attack of scurvy was only one manifestation.

**Treatment.**—This is remarkably simple—viz., to discontinue all proprietary foods, condensed milk or sterilised milk, and to substitute a diet of fresh cow's milk, modified to suit the child's digestion. With this treatment alone improvement will soon begin and gradually complete recovery takes place. However, when fresh fruit juice is added improvement is much more rapid. It should always be combined with the change in diet. Orange juice is to be preferred, but the juice of any fresh ripe fruit will answer the purpose. Oranges should be sweet and fresh. From two to four ounces a day are required, best in divided doses, given about one hour before the milk-feeding. It may be given plain, or diluted with water. In some cases, when not well tolerated by the stomach, it is better given at night, when no food is taken. Potato also has marked anti-scorbutic properties, and may be given in the form of a purée to infants as young as eight or ten months. The only really difficult cases to manage are those in which the general condition approaches one of marasmus, or when scurvy is accompanied by marked gastric or intestinal disturbance. When an intestinal catarrh is present, with the bowels moving five or six times a day, one may hesi-

tate to give the fruit juice for fear of increasing these symptoms. In a number of instances I have seen intestinal symptoms, which had resisted ordinary measures, immediately improved by the fruit juice, thus establishing their intimate connection with the scorbutic condition.

Other things of value are fresh beef juice, and for older children all fresh vegetables, especially potato. The anæmia and malnutrition call for iron, cod-liver oil, and other tonics, which should be given after active symptoms of the disease have disappeared. Infants with scurvy should be handled as little as possible, and should be particularly protected against exposure in their extremely susceptible condition. The affected limbs should be immobilised by splints during the period of marked symptoms, always if epiphyseal separation has taken place, and in many other severe cases.

#### RICKETS (*Rachitis*).

Rickets is a chronic disease of nutrition. While the only important anatomical changes are found in the bones, it is not to be regarded as a bone disease; but as a very complex pathological process, the result of disturbed metabolism, which affects chiefly the bones, but also muscles, ligaments, mucous membranes, and nearly all the organs of the body, particularly the nervous system. It occurs especially between the ages of six and eighteen months. It is not very common in the country, but is exceedingly frequent in most large cities. While not a fatal disease *per se*, rickets adds very greatly to the danger from all acute diseases in infancy, and even to some degree also to those of later life. Under proper conditions of diet and hygiene it tends to spontaneous recovery.

**Etiology.**—Certain facts in the causation of rickets are well known. It is closely related to improper feeding and bad hygienic surroundings. It is not common in nursing children unless lactation is unduly prolonged,<sup>1</sup> as, for example, where nursing is continued for fifteen to eighteen months without other food. Artificially fed children are much more prone to the disease, especially those who are badly fed. The diet in these cases is most frequently deficient in fat, and often at the same time in protein, while it is apt to contain an excess of carbohydrates. It is somewhat difficult to separate the effects which these different factors produce. It appears, however, that the most important factor is the deficiency in fat. Rickets is exceedingly common in children reared upon the proprietary foods, nearly all of which are very low in fat and contain an excess of carbohydrates. It is also common in children who are reared upon sweetened condensed milk, and for precisely

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<sup>1</sup> An exception to this statement must be made in the case of Italian and Negro children. In this class as observed in New York it is not uncommon to see marked rickets in those getting nothing but the breast.



the same reason. When both fat and protein are low, rickets is more likely to result than when only the fat is deficient.

Certain experiments have been made which show that a condition of the bones resembling rickets may be produced in animals by a diet deficient in calcium salts, and furthermore that this may be cured simply by the addition of these salts to the food. The conclusion can not, however, be drawn that rickets in children is produced in this manner. In the first place the bony condition in the artificial disease is not histologically the same as that seen in rickets; again, rickets in the child is not cured simply by the administration of calcium salts; and, finally, rickets develops where these elements have not been deficient in the food.

Hygienic surroundings are next in importance to diet. Although, as previously stated, rickets is essentially a disease of cities, being most often seen in children living in crowded tenements where the effects of improper food are most strikingly shown, yet even here the disease is rare in those who get a plentiful supply of good breast-milk.

*Distribution of Rickets.*—According to Palm, the disease is almost unknown in the extreme north—Greenland, Iceland, Norway, and Denmark. It is also very rare in China, Japan, Greece, Turkey, and the southern portions of Italy and Spain. Its greatest frequency is in the temperate zone. The general immunity of children in southern latitudes appears to be due to the out-of-door life, and the almost universal custom of maternal nursing. In the cities of America no race is exempt from the disease. In New York the greatest susceptibility is among the Negroes and the Italians. The extreme cases of rickets seen are almost invariably in one of these nationalities. It is exceptional to see in a dispensary or hospital a child of either of these races who does not show, to a greater or less degree, the signs of rickets. These two southern races seem to bear very badly the climate and the confined life of the northern cities. So far as my observations are concerned, there is no peculiarity in the food of these people which explains the prevalence of rickets among them, and this must be attributed to a race peculiarity. In the country, the immunity from rickets is due partly to the more prevalent custom of maternal nursing, and partly to the better surroundings; the increased resistance of the children rendering them much less susceptible to the influences of bad feeding than is seen in the cities. In New York among dispensary and hospital patients, rickets is exceedingly common, and is seen chiefly in the foreign elements of the population.

*Heredity.*—There is no evidence that rickets is hereditary. Any cachexia in the parents, such as syphilis, tuberculosis, or alcoholism, may, however, by diminishing the child's resistance, be a predisposing cause of rickets. The later children in a family are more likely to be affected than the earlier ones, especially when the interval between the pregnancies has been short.

*Previous Disease.*—Rickets not infrequently develops in syphilitic children; the connection, however, seems to be no closer than with any other cachexia. Chronic disorders of the digestive tract sometimes precede and often follow the development of rickets. There is no sufficient ground for believing that rickets exerts any protective influence against tuberculosis; on the contrary, the thoracic deformity of rickets may be a predisposing cause.

Rickets affects both sexes with equal frequency. The symptoms usually manifest themselves between the sixth and eighteenth months. Congenital and late rickets will be considered separately.

*Nature of the Disease.*—Rickets is a disorder of nutrition, the result of some disturbance of metabolism in which calcium plays a very important rôle. The exact nature of this disturbance is not yet understood. Three theories have been advanced in explanation of the deficiency of calcium in the bones which is the only constant lesion of the disease. The first one, that rickets is due to a lack of calcium in the food, is not supported either by clinical or experimental evidence. The second theory is that the disease is due to an increased excretion of calcium as a result of disturbances of digestion. The frequent occurrence of rickets after prolonged disturbances of digestion lends some support to this view. The third theory advanced is that although sufficient salts are furnished in the food, they are excreted in excess because the bones are incapable of assimilating them.

*Lesions.*—The only constant and characteristic lesions of rickets are found in the bones; these changes are sufficiently definite to give it a place as an essential disease and not merely a form of malnutrition. It is still a matter of dispute whether these bony changes are to be considered as inflammatory, or simply as the result of disordered nutrition. Disordered nutrition and chronic inflammation are closely allied, and it really makes but little difference which view is taken. Occurring at a time when the growth of bone is so rapid, the effects of rickets are very striking and very serious.

In order to appreciate how the bones are affected by rickets, it must be remembered that the long bones grow in length by the production of bone in the cartilage between the epiphysis and the shaft; that the shaft grows in thickness by the production of bone beneath the inner layer of the periosteum; and that the medullary canal is continually increasing in size by the absorption of the inner layers of the bone. In rickets there is an exaggerated production of cartilage at the epiphysis, and excessive cell-growth beneath the periosteum, while the process of ossification in these tissues goes forward slowly and imperfectly, or is entirely arrested. At the same time the absorption of the medullary layers may be even more rapid than normal. In health the growth of bone in length is much more rapid than its increase in diameter, owing to the greater activity

of the changes taking place at the epiphysis; so, in rickets, it is at the extremities of the long bones that the most marked changes are seen.

One of the most striking features of rachitic bones is their unnatural flexibility. This is due to deficient ossification in the superficial layers of the shaft of the long bones, and also at their extremities. Normally, bone contains about one-third organic and two-thirds inorganic matter. In marked rickets the proportions are reversed, the bones often containing twice as much organic as inorganic matter. Changes are seen in all the long bones, but all are not affected to the same degree. Sometimes those most affected will be the bones of the leg, sometimes those of the forearm, and sometimes the ribs. The extent varies with the severity of the process.

There are characteristic changes in form. The most constant is enlargement of the epiphyses of all the long bones. This is most strikingly seen in the lower extremities of the radius and tibia. The enlargement may be so marked that the width of the epiphysis is increased by one-half. All the sharp angles, borders, and prominences of the bones are rounded off. The curvatures of rachitic bones are more fully described under the head of Symptoms. They may be due to a variety of causes. Some are simply an exaggeration of the normal curves, much increased by the swelling of the epiphyses; others are due to muscular action, to atmospheric pressure, to some unnatural posture, such as the cross-legged position, to the weight of the limbs, or to the weight of the body. The principal change in the form of the flat bones consists in the production of large bosses or prominences due to thickening of the bone, usually about the centre of ossification. These bosses are soft and spongy. Fractures are not uncommon. The bones most frequently broken are the radius and ulna; next, the clavicle, the ribs, the humerus, and the femur. The fractures are usually of the green-stick variety. There is a bending of the outer and a fracture of the inner layers of the shaft of a long bone. This results in more or less impaction, and is usually followed by the production of considerable callus. The epiphyseal changes result in arrested growth in length, rachitic bones being usually much shorter than normal. Increased vascularity is seen in the bosses upon the flat bones, at the extremities of the long bones and upon stripping the periosteum from the shaft.

In a longitudinal section of one of the long bones, the principal change seen at the extremity is that the cartilaginous layer which unites the epiphysis and the shaft is very much enlarged, both in width and thickness, the latter being sometimes four or five times the normal. This cartilaginous area is of a bluish colour, rather softer than normal cartilage. On one side it blends with the cartilage of the epiphysis, on the other it presents an irregular dentated border, and in it the calcified areas are irregular and scattered. The epiphyseal centres of ossification

are enlarged, softer, and more vascular than normal, thus increasing the size of the extremity of the bone. In the shaft, the outer layers of bone are thickened and soft, like decalcified bone, the deeper parts being firmer, while the deepest layers may be completely ossified. The medullary canal is much more vascular than normal, its contents resembling granulation tissue. Toward the extremities the trabecular spaces are much increased in size, so that the bone appears unnaturally porous. On vertical section of one of the flat bones—e. g., one of the bosses upon the skull—there is found a great increase in the size of the trabecular spaces. The bosses are made up of large spongy masses, so soft as to be easily indented with the finger.

*Microscopical Changes.*—At the junction of bone and cartilage at the extremity of one of the long bones, there are readily traced in normal bone (Fig. 34) several distinct zones. Next to the hyaline cartilage (*a*) there is a proliferating zone (*b*), made up of cartilage cells and matrix, the cells having no orderly arrangement. Next to this is a columnar zone (*c, d*), in which the cartilage cells are arranged in regular rows or columns. Adjoining this is the zone of calcification (*e*); and, finally, there is the zone of ossification (*f, g*), where true bone is formed.

In rickets (Plate IV and Fig. 35), the principal changes are seen in the proliferating and columnar zones. The proliferating zone (Fig. 35, *b*) is increased chiefly by the multiplication of new cells; it is also more vascular than normal. The columnar zone (*c*) is affected in a similar way and to a much greater degree. It is less regular in its formation, and, instead of containing but few vessels, it shows large vascular channels, sometimes surrounded by medullary spaces (*e*). The ossification zone, instead of being narrow and sharply outlined, is broad and very irregular. Calcified areas (*f*) may be seen in the midst of regions which are cartilaginous, while masses of cartilage (*h*) occupy areas which should be completely calcified. In some places there appears to be a transformation of cartilage into bone-tissue of an inferior sort by a direct or metaplastic process. In the shaft there is seen more or less thickening, and an increased vascularity of the periosteum. Beneath the inner layer there is excessive cell-proliferation, while calcification of this new tissue is imperfect or absent, and instead of hard, compact bone, we find irregular, spongy masses. In the spongy bone there is considerable thickening, with an erosion of bony trabeculae, which results in the formation of large medullary spaces filled with blood-vessels and connective tissue rich in cells.

*Termination of the Rachitic Process.*—After a variable time, usually from three to fifteen months, the active proliferative process going on in the cartilage and beneath the periosteum ceases, and is gradually replaced by ossification. The bone becomes less vascular, and a rapid formation of bone takes place in the normal way. In addition, there is in some

places a direct transformation of cartilage into bone. Condensation and contraction take place in the spongy masses of bone. As the result of this, the affected bone may become even harder than normal; often it is ivory-like. Its structure, however, is never quite like that of healthy bone.

In the long bones the epiphyseal swellings slowly diminish, and may quite disappear; the slighter curvatures may be entirely overcome, and

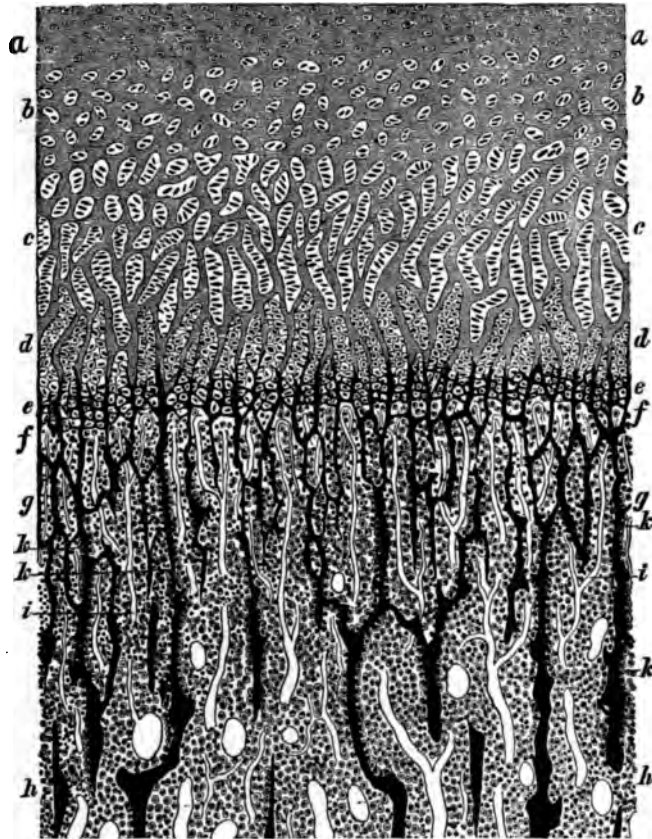
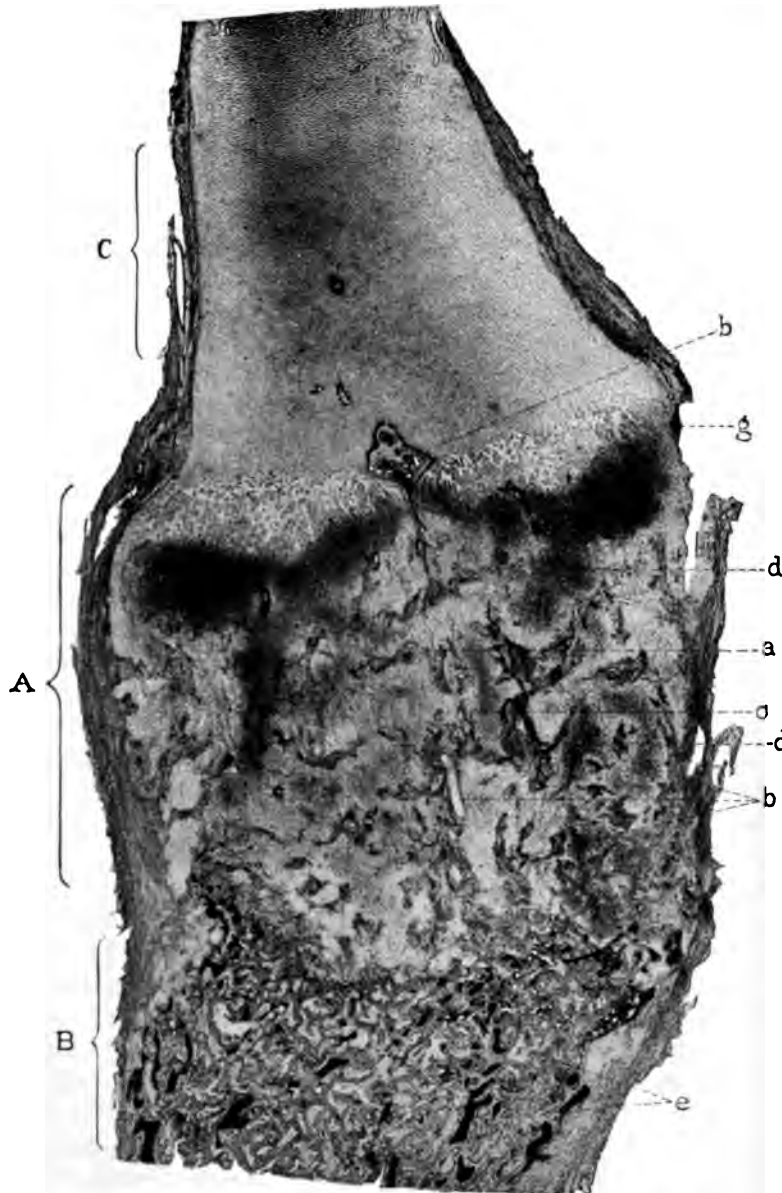


FIG. 34.—SECTION THROUGH OSSIFICATION ZONE OF NORMAL BONE (Ziegler). *a*, hyaline cartilage; *b*, zone of beginning cartilage proliferation; *c*, columns of cartilage cells; *d*, columns of hypertrophic cartilage; *e*, zone of temporary calcification; *f*, zone of primary medullary spaces; *g*, zone of primary bone formation; *h*, fully developed spongy bone; *i*, blood-vessels; *k*, layer of osteoblasts.

the greater ones much lessened. The beading of the ribs becomes almost imperceptible; the bosses upon the skull shrink very markedly, and may leave scarcely a trace of their existence. In most cases the active process in rickets comes to an end by the time the child is two and a half years old, often at two years.

PLATE IV.



BONE IN RICKETS.

Longitudinal section of a rib at the junction of the costal cartilage, in a severe case of rickets (slightly magnified). C = costal cartilage, B = bone, A = proliferating cartilage-zone, which is much widened. Between the hypertrophied cartilage cell-columns (a) making up this proliferating zone, are seen medullary spaces (b) containing blood-vessels. In this zone lie masses of bone (c) not calcified. The calcification zone is almost wanting, only scattered islands (d) of calcified cartilage-cells being seen. Beyond this proliferating zone (A) is a layer of bony tissue (B) made up of small bands of which only a few have a nucleus containing lime (e). These nuclei appear black. The bony bands differ both in form and arrangement from those of normal ossification. Between the bony masses are medullary spaces which appear light in the illustration. At (g) the beginning of cartilage proliferation is seen. Above this zone the cartilage is normal.

(From Karg and Schmorl.)



*Visceral Lesions.*—These are not infrequent, but are not essential to rickets. In the lungs they are due to deformities of the chest wall and



FIG. 35.—RACHITIC BONE (Ziegler). LONGITUDINAL SECTION THROUGH OSSIFICATION ZONE OF THE UPPER DIAPHYSIS OF THE FEMUR OF A MODERATELY RACHITIC CHILD ONE YEAR OLD (highly magnified). *a*, unchanged hyaline cartilage; *b*, beginning cartilage proliferation; *c*, columns of proliferated cartilage cells; *d*, columns of proliferated hypertrophic cells; *e*, medullary spaces containing blood-vessels lying within the cartilage; *f*, calcified cartilage; *g*, bony tissue; *h*, remains of cartilage within the bony tissue; *i*, point of uncalcified bony tissue; *k*, calcified bony tissue.


to complications. Beneath the deep lateral furrows which are so common, there is found a part of the lung in a state of more or less complete collapse. This is accompanied by emphysema of the portion just ante-



rior to it. Acute and chronic bronchitis and broncho-pneumonia are exceedingly frequent. A low grade of chronic catarrhal inflammation of the stomach and intestines is common, and is often associated with dilatation of these organs. The spleen is enlarged in most cases during the period of active symptoms. This is usually moderate in degree, although marked enlargement is not at all rare. The swelling of the spleen is chiefly due to simple hyperplasia. Enlargement of the liver is less frequent, and may occur with or without that of the spleen. There are no constant changes in the structure of these organs. The lymph nodes are frequently enlarged. Rachitic patients are more prone to these swellings than are other children. They are due to simple hyperplasia, and have no close connection with rickets. Cerebral changes are rare, and those described are rather of accidental occurrence than dependent upon the rachitic process. As stated under Symptoms, enlargement of the head is usually due to thickening of the cranial bones. Although hydrocephalus is occasionally seen, it is extremely doubtful whether it is more frequent than in patients not rachitic. Hypertrophy of the brain has been described in connection with rickets, but as yet this does not seem to be established by sufficient pathological evidence. The muscles are flabby from imperfect nutrition, and sometimes atrophied from disuse, but no essential anatomical changes have been demonstrated in them.

**Symptoms.**—A well-marked case of rickets makes a striking picture (Plate V), and one not easily mistaken. There are seen the large head, beaded ribs, narrow chest, prominent abdomen, symmetrical swellings of the epiphyses of the wrists and ankles, and curvatures of the extremities. The beginning of symptoms is nearly always insidious, and the patient does not usually come under observation until they have existed for several weeks, often several months.

**EARLY SYMPTOMS.**—The most constant early symptoms are sweating of the head, extreme restlessness at night, constipation, beading of the ribs, and cranio-tabes. The head-sweating is rarely absent, and may continue for several months. It is especially profuse during sleep, the perspiration standing out in large drops upon the forehead, often being sufficient to wet the pillow. This is one of the causes of the nasal and bronchial catarrhs so common in rachitic infants. There is marked restlessness during sleep: the children tossing about the crib, kicking off the clothes, and never having the quiet, natural slumber of healthy infants. This may be due to many causes, but when persistent and associated with marked perspiration of the head, rickets should be suspected. In many rachitic infants more serious nervous symptoms appear early; there may be tetany, laryngismus stridulus, or general convulsions. Constipation is frequently seen as an early symptom, although it is more marked in the later stages of the disease.



**PLATE V.**



**TYPICAL RICKETS.**

**Showing the large head, narrow chest, prominent abdomen, marked enlargement of the epiphyses at the wrists and ankles. There are also curvatures of the forearms and legs which are not so well shown.**

**The patient a child two and a half years old.**



The beading of the ribs is almost invariably the first appreciable change in the bones, and it is well-nigh constant. This forms the so-called "rachitic rosary," consisting of nodules at the line of junction of the costal cartilages and the ribs. It may be slight, or there may be a row of knobs as large as small marbles. In many cases with marked thoracic deformity, little or no beading of the ribs is seen externally,



FIG. 36.—RACHITIC SKULL. From coloured child two years old, horizontal section, inner surface; showing thickening of the bones, especially the frontal, and open fontanel.

although at autopsy it is found to be very marked upon the internal surface of the chest (Plate VI). Beading of the ribs was noted in all but two of one hundred and forty-four successive cases of rickets, at the time of the first examination. In infants under six months there may be found soft spots in the cranium, usually over the occipital or posterior portions of the parietal bones. These are from one-fourth to one inch in diameter, and there are usually several of them present. By pressure with the finger they give a sort of parchment-crackling sensation. This condition is known as cranio-tabes. Cranio-tabes is believed to be more frequent when syphilis is associated with rickets, and it is seen also in syphilitic cases which are not rachitic. A rachitic cachexia is not usually

present until the symptoms have existed for several months, and in many cases it is not seen at all.

**DEFORMITIES.**—The deformities of rickets are almost invariably symmetrical in character, and usually numerous. In extreme cases almost every bone in the body is affected.



FIG. 37.—RACHITIC HEAD. Italian child two years old; square, prominent forehead and flat vertex.

**Head.**—This usually appears to be too large, and although it may not be greater in circumference than that of a healthy child of the same age, it is out of proportion to the rest of the body. In marked cases the increase in circumference may be one or two inches. The enlargement is chiefly due to thickening of the cranial bones. In one case with marked deformity, I found the skull over the parietal bones half an inch in thickness (Fig. 36). This thickening diminishes with recovery, but in most cases the head remains throughout life larger than it



FIG. 38.—RACHITIC SKULL FROM A CHILD ONE YEAR OLD. Showing frontal and parietal bosses and wide fontanel.

should be. The shape of the rachitic head is somewhat square (Fig. 37), owing to the formation of large bosses over the parietal and frontal eminences. It is flattened at the occiput from pressure, and flattened also at the vertex. In extreme cases, the prominences upon the frontal and parietal bones may be so great as to produce quite a marked furrow along the line of the sagittal and frontal sutures, and one at right angles to this along the coronal suture (Fig. 38). This condition gives unusual prominence to the forehead. Marked deformity of the head has been observed in thirty-three per cent of my cases. The sutures may remain open for an unnatural time, occasionally until the end of the first year. The fontanel is late in closing, being frequently found open at two and a half, and sometimes even at three years. Often at eighteen or twenty months the fontanel is two inches in diameter. The veins of the scalp are often prominent, and the hair is frequently worn from the occiput, owing to restlessness during sleep. Occasionally rickets and hydrocephalus are associated, but the latter is the least frequent of all causes of the enlargement of the head.

*Chest.*—Beading of the ribs has already been mentioned. This is the most characteristic feature, but in the majority of cases there are, in

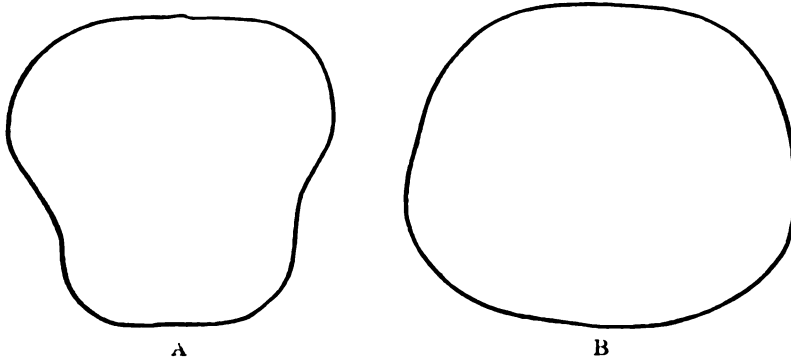


FIG. 39.—A, HORIZONTAL SECTION OF A RACHITIC CHEST, child two years old, showing lateral furrows; B, SECTION OF CHEST OF HEALTHY CHILD OF THE SAME AGE.

addition, lateral depressions over the lower third of the chest, at the line of junction of the cartilages with the ribs, with eversion of the lower borders of the ribs. In severe cases these depressions or furrows are so great as to cause serious deformity (Plate VI). Usually there is a great diminution in the transverse, and an increase in the antero-posterior, diameter of the chest. Fig. 39 shows the outline of the chest of a rachitic child of two years, compared with that of a healthy child of the same age. Another frequent deformity is the "rachitic girdle," which consists in a transverse depression about two inches broad, extending from one side of the chest to the other, just above its lower border. A less

frequent deformity is the "funnel chest," a deep central depression over the ensiform cartilage. This is sometimes nearly an inch and a half in depth. Marked thoracic deformity was seen in twenty per cent of my cases, and in only a small proportion was the chest normal.

The factors in the production of the thoracic deformity are the contraction of the diaphragm, atmospheric pressure, and soft chest walls, these sinking in at the point where they have least resistance, viz., at the junction of the costal cartilages and the ribs. When there exists any obstruction to the entrance of air, as with bronchitis, hypertrophied tonsils, or adenoid growths of the pharynx, the thoracic deformities are exaggerated. Irregular chest deformities depend upon the co-existence of pathological conditions in the lungs. Pigeon-breast is occasionally seen, but it is doubtful if this depends upon rickets alone.

*Spine.*—In very many of the milder cases this is normal. The most characteristic deformity consists in a posterior curve (kyphosis), (see Fig. 40), which is a general one, usually extending from the mid-dorsal to the sacral region. This existed in forty-six per cent of my cases. In the early part of the disease it disappears entirely on suspending the child, or making extension upon the extremities; but in cases of long standing it may not disappear entirely by these tests. Very much less frequently there is seen a rotary curvature. This, in my experience, has been more frequently with the convexity to the left side than to the right—the opposite of the common form of lateral curvature seen in young girls. Marked lateral curvature in children under three years is usually rachitic.



FIG. 40.—RACHITIC CURVATURE OF THE SPINE.

The clavicle is affected only in severe cases. The usual deformity consists in an exaggeration of the anterior curve at the

inner third of the bone, which is somewhat shortened and its extremities enlarged. It is not infrequently the seat of green-stick fracture.

Deformities of the pelvis belong to obstetrics rather than to pædiatrics. The most common rachitic change is a diminution of the antero-posterior diameter and a narrowing of the subpubic arch.

*Extremities.*—Deformities of the upper extremities are usually symmetrical. The humerus is affected only in severe cases. It has a forward and outward curve, although rarely a very marked one. Both the epiphyses are enlarged, although the upper one can not well be made out unless the child is very thin. The radius and ulna are frequently affected. They present a convexity upon their extensor surfaces (Plate

**PLATE VI.**



**DEFORMITY OF THE CHEST IN SEVERE RICKETS.**

**In the upper picture, giving the external view, is shown a deep oblique furrow at the junction of the ribs and costal cartilages, these meeting at an acute angle.**

**In the lower picture the ribs have been separated from the spine and spread open, showing the same deformity as it appears from within, looking forwards.**

**From a coloured child ten months old.**





V), which in some cases is very marked, particularly in children who have been creeping. Green-stick fractures here are quite frequent



FIG. 41.—MULTIPLE FRACTURES IN RICKETS.

Showing both arms of the same patient; fractures also of both femora.

as they are also in the femora. They are frequently multiple and occur from very slight causes, sometimes apparently from muscular contraction. Cases with such fractures are sometimes classed as osteomalacia. Rachitic changes at the epiphyses are more common than in the shaft, enlargement of the epiphyses at the wrist being one of the most constant bony deformities of rickets (Plate V). It was present in ninety-five per cent of my cases. Less frequently similar swellings are seen at the elbow. Enlargement of the ends of the metacarpal bones or the phalanges I have seen but seldom and only in extreme cases.

The lower extremities are rather more frequently affected than the upper, but in a similar way. The femur is involved only in severe cases; it commonly presents a general forward and outward curve, which is mainly due to the weight of the legs as the child sits. Occasionally there is also an outward rotation of the femur, where children have been allowed to sit much in a cross-legged posture. When such children begin to walk, the toes are turned very far outward. The principal deformities



FIG. 42.—TYPICAL BOW-LEGS OF SEVERE FORM.

of the lower extremity are bow-legs (Fig. 42) and knock-knees (Fig. 43). Knock-knees are more common in females, and are believed to be due to an overgrowth of the inner condyles of the femur. Enlarge-



FIG. 43.—KNOCK-KNEES.

ment of both condyles can be demonstrated in most of the marked cases of rickets. The cases of slight bow-legs may be due simply to swelling of the epiphyses, the shaft of the bone being quite normal. This point I have verified by post-mortem observations. Such are probably most of the deformities which disappear spontaneously. The most severe cases of bow-legs are often associated with some degree of anteroposterior curvature, and the latter may be the principal deformity. Enlargement of the epiphyses at the ankles is usually present when it is seen at

the wrists, and nearly to the same degree. Enlargement of the upper epiphyses of the tibia and the fibula is seen only in severe cases. The cause of the deformities of the leg is not, primarily, at least, walking too early, since they are common in children who have never walked; slight deformities, however, may be aggravated by early walking. A change which has not been sufficiently emphasised is the arrested growth of the long bones; this is one of the most characteristic features of rickets. A rachitic child of three years often measures in height five or six inches less than a healthy child of the same age, the difference being almost entirely in the lower extremities.

All the *ligaments*, but particularly those about the large joints, are lax and frequently elongated. This may lead to the deformity known as weak ankles, or to an over-extension at the knee (*genu recurvatum*); also to unnatural mobility at the hips, shoulders, elbows, and wrists. The condition of the ligaments plays an important part in the production of spinal deformities.

**MUSCLES.**—The muscular symptoms of rickets are almost as constant and as characteristic as those of the bones. The muscles are small, very flabby, and poorly developed; hence rachitic children are unable to sit

erect, or to stand or walk at the proper age. Of one hundred and fifty-one cases in which the date of walking alone was investigated, only twenty-seven, or eighteen per cent, walked before the fifteenth month; forty-seven per cent were not walking at the eighteenth month; twenty per cent, not at two years; and ten per cent, not at two and a half years. Late walking is one of the most common symptoms for which advice is sought by parents with rachitic children. The muscular power in the extremities is sometimes so feeble as to suggest paralysis. I have seen a number of cases in which the symptoms so resembled paralysis, that even expert diagnosticians were unable to differentiate rickets from poliomyelitis except by the electrical reactions, those in rickets being usually normal or exaggerated. In other cases the symptoms may suggest cerebral palsy of the flaccid type. The muscular symptoms may be marked when the bony changes are slight, and conversely. As no lesions of the muscles have been demonstrated, the symptoms are probably due to imperfect nutrition. Two other symptoms depend chiefly upon the condition of the muscles, viz., pot-belly and constipation.

Pot-belly is quite an early symptom, and in most cases a very marked one (Plate V). It was noted in sixty per cent of my cases. The enlargement of the abdomen is uniform. It is everywhere tympanitic, and it may be as tense as a drumhead. It is due to a loss of tone in the abdominal muscles, and in the muscular walls of the stomach and intestine. It is aggravated by chronic indigestion and excessive intestinal putrefaction. The enlargement is thus mainly from tympanites. There may be a marked degree of dilatation both of the stomach and the colon. To a very small degree only, does the large abdomen depend upon swelling of the liver or spleen.

The constipation of rickets, as already suggested, depends upon the loss of tone in the muscular walls of the intestines. It may alternate with diarrhoea. It rarely happens that a rachitic child has habitually normal evacuations from the bowels. Hard, dry, constipated stools frequently set up a condition of chronic catarrh of the colon in which large masses of mucus are discharged.

**FEVER.**—According to some observers there is a febrile movement which belongs to the active stage of rickets, but I have never been able to satisfy myself of the truth of this observation.

**DENTITION.**—As a rule, dentition is late and apt to be difficult, i. e., it is associated with attacks of indigestion or other disturbances which may be serious. Individual cases, however, present great variations in regard to this symptom. A study of the progress of dentition in one hundred and fifty rachitic children gave the following results: in fifty per cent the first teeth were cut on or before the eighth month; twenty per cent of the cases had no teeth at twelve months, and in eight per cent none had appeared at fifteen months. Even though the first teeth

come at the usual time, the progress of dentition is usually retarded by the development of rickets. The character of the teeth in rickets is usually good. This is in striking contrast to hereditary syphilis, where the tendency to early decay is constantly seen.

**GENERAL APPEARANCE.**—Children suffering from marked rickets are almost always anæmic. The majority are fat and flabby. The tissues are soft and have but little resistance. Rarely, they may be thin, like patients suffering from marasmus.

Rachitic patients are very prone to suffer from hypertrophied tonsils, adenoid growths of the pharynx, and enlargements of the lymph nodes of the neck. In all forms of acute illness the feeble resistance of these patients is very evident. This is especially true of acute disease of the lungs.

The *mucous membranes* are very vulnerable in all rachitic patients. From the slightest indiscretion in diet an attack of acute indigestion or diarrhoea may be brought on, and from a very insignificant exposure, catarrhal inflammation of the upper or lower air passages is excited. In rachitic patients all such attacks are prone to run a protracted course. Inflammation of the trachea and larger bronchi is likely to extend to the smaller bronchi and the lungs.

The downward displacement of the *liver* and *spleen* from contraction of the chest should not be mistaken for enlargement of these organs. Moderate enlargement of the spleen is very common during the stage of most active symptoms, i. e., from the sixth to the twelfth month. Great enlargement of either liver or spleen is infrequent.

**BLOOD.**—Anæmia is present in most of the marked cases, its intensity varying with the severity of the rachitic process. The blood picture is usually that of an ordinary secondary anæmia. Leucocytosis is often present; it is more marked in cases attended by an enlarged spleen.

**NERVOUS SYMPTOMS** are among the most frequent manifestations of rickets. Restlessness at night has already been mentioned as a prominent early symptom. Pain and tenderness are rare. A disposition to muscular spasm is seen in many cases. There may be laryngismus stridulus, tetany, or general convulsions. While in all infants, owing to the irritability of the nervous centres, convulsions are easily excited from relatively slight causes, in those who are rachitic this susceptibility is greatly intensified. As a predisposing cause of convulsions in infancy, rickets takes the first place. The younger the child and the more active the rachitic process, the more frequently do convulsions occur. They belong especially to the first year, being most frequent between the third and sixth months. The exciting cause of convulsions in these cases is usually to be found in the stomach or intestine.

**Course and Termination.**—Rickets is essentially a chronic disease, and its course is measured by months. The active symptoms in most cases

continue from three to fifteen months. That active symptoms cease when a child reaches the age of eighteen months or two years, is no doubt due largely to the fact that at this age the diet is more general, and is more likely to furnish what the child needs, and that more fresh air is likely to be secured than at an earlier age.

The earliest symptoms of improvement are a diminution in the nervous symptoms, especially in the restlessness at night; increased muscular power, as shown by a disposition to stand or walk; diminution in the head-sweats; disappearance of the cranio-tabes; and improvement in the anæmia. The changes in the deformities are very slow, and from month to month almost imperceptible. When improvement once begins, however, it usually goes steadily forward.

**Types of Rickets.**—*Congenital Rickets.*—Infants may present at birth the characteristic deformities of rickets, and there may be found even the minute bone changes of the disease. Such cases are reported to be common in Vienna and other large cities of Europe, where mothers during pregnancy have lived under unfavourable conditions. In America, however, congenital rickets is a very rare disease. Single cases have been reported by several writers; but it must be remembered that cretinism and chondro-dystrophy have often been improperly included under this head.

*Late Rickets.*—Rare instances have been reported of bony deformities in all respects like those of rickets, developing in children from six to twelve years old. A number of such cases have been observed in England. I have not seen this disease, nor has a case been seen during the past twenty years at the Hospital for Ruptured and Crippled, New York, where more deformities come under observation than anywhere else in this country.

*Acute Rickets.*—Although from time to time cases have been reported with this title, from a study of the histories it is clear that the great majority, if not all of them, were cases of infantile scurvy. It is doubtful whether, strictly speaking, there is such a thing as acute rickets.

**Diagnosis.**—The diagnosis of rickets is not usually difficult. The most important early symptoms for diagnosis are sweating of the head, cranio-tabes, great restlessness at night, delayed dentition, and enlarged fontanel. All these, taken separately, may mean something else, but collectively they can mean nothing but rickets. In the later stages some of the characteristic deformities are usually present; the most constant are beading of the ribs, enlargement of the epiphyses of the wrists and ankles, and bow-legs.

Special symptoms, when unusually prominent, may give rise to difficulty in diagnosis. The enlargement of the head may be mistaken for hydrocephalus. The delayed dentition and large fontanel of the cretin may be mistaken for rickets. Muscular weakness may be so great, espe-

cially when affecting the legs, as to make it easy to mistake a rachitic pseudo-paralysis for actual paralysis due to a cerebral or spinal lesion. When walking is much delayed, rickets may be passed over as simple backwardness. In nearly all of the last-mentioned group of cases the diagnosis may be cleared up by a careful search for the bony changes, and by the fact that in rickets there is only a general weakness of all the muscles, and not actual paralysis of any limb or group of muscles. The greatest difficulty is usually found where the muscular symptoms are marked and the bony changes slight, as is not infrequently the case. Here the question is, whether rickets is sufficient to explain all the symptoms, or whether in addition some other condition is present. The electrical reactions will decide the question of poliomyelitis, while the presence of cerebral symptoms, exaggerated knee-jerks, and rigidity of the legs, will usually mark a cerebral birth-palsy. The bony enlargements of syphilis may be confounded with those of rickets. The bone changes of early syphilis, although affecting the epiphyses are seen at an earlier age and are generally accompanied by pain and tenderness, sometimes by epiphyseal separation, none of which are seen in rickets. The bone changes of late syphilis affect the shaft rather than the extremities of the long bones; where the bone is enlarged near the joint it is usually upon one side only. In syphilis there may be necrosis, while in rickets breaking down of bone is never seen. From scurvy, rickets is differentiated by the absence of marked hyperæsthesia, ecchymoses, and other hæmorrhages, the changes in the gums, and most of all by the fact that anti-scorbutic diet produces no immediate change in the symptoms. The diagnosis of rachitic curvature of the spine from vertebral caries will be considered in connection with the latter disease.

**Prognosis.**—Rickets *per se* is seldom, if ever, a cause of death. It is, however, a large factor in the mortality of the first two years, as it predisposes strongly to many forms of acute disease. It is an important etiological factor in certain serious nervous conditions, especially convulsions. Rickets adds very greatly to the danger from all acute diseases of infancy, particularly those of the respiratory tract. The encroachment upon the capacity of the lungs by a marked thoracic deformity, may in itself be enough to keep a child in a delicate condition and retard its growth. At the same time such a condition is a constant invitation to acute attacks of bronchitis or pneumonia. The effect of rickets upon the future health of the child depends chiefly upon the presence and extent of the thoracic deformity. When this is absent, although children may remain somewhat dwarfed on account of their short legs, in other respects they may be as well as if they had never been the subjects of rickets.

**Prophylaxis.**—As rickets is primarily due to improper food or feeding, and secondarily to bad surroundings, it may largely be prevented

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by the observance of proper rules of feeding as laid down elsewhere, and by removing children from their faulty surroundings. Especial care should be given to the later children of a family where the earlier ones have shown even the mildest symptoms of rickets, as the predisposition is sure to increase with each successive child.

**Treatment.**—In considering the treatment of rickets, the natural course of the disease is to be kept in mind, viz., that active symptoms frequently continue only until the end of the first year, rarely longer than the eighteenth or twentieth month. The most important period for treatment, therefore, and the one in which it is most effective, is from the sixth to the eighteenth month. The earlier the treatment is begun the better will be its results. General treatment after the eighteenth month, has very little effect upon the disease, for by this time most of the harm has been done. The course of the disease when untreated is toward spontaneous recovery, from the changes in diet and life which are usually made when children have reached the latter half of the second year. Most of the cases seen in private practice are of a mild type and recover without special treatment, often no diagnosis being made until later in life, when the bony deformities or stunted growth indicate the previous existence of rickets. The first step in treatment is to remove the cause, and is therefore to be directed to the diet and hygiene of the patient. The results will depend upon how completely these causes can be discovered and removed.

**Diet.**—Such disorders of digestion as are present must be treated on general principles. The most frequent dietetic error in rachitic patients being an excess of carbohydrates and an insufficient supply of fat, it follows that condensed milk, proprietary infant foods, and large amounts of farinaceous foods of every description should be stopped. A suitably modified cow's milk should be substituted or for young infants a wet-nurse should be secured. Most infants, however, are eight to ten months old before rachitic symptoms are observed; to them beef juice, raw eggs, and fruit juice should be given in addition to milk. Cream, though desirable, is very often badly borne and some other form of fat must be substituted. For many infants olive oil will be found useful and may be given, one teaspoonful three times a day for long periods. The fat of crisp bacon upon stale bread or zwieback among the poor may serve as well. All these articles are to be given according to the rules laid down in the chapters on Infant Feeding.

**Hygiene.**—In large cities it is almost impossible to secure for rachitic patients the surroundings they require. Whenever possible, such children should be sent to the country; but where this is out of the question, much may be accomplished by frequent excursions upon the water or into the country, by keeping children as much as possible in the parks and open squares of the city, and securing plenty of fresh air in sleeping



rooms. Cold sponge-baths given every morning, do much to lessen this susceptibility. Sunshine, though difficult to obtain in large cities, is a most efficient therapeutic agent. The establishment of suburban hospitals and homes for these cases would do more than anything else to lessen the mortality from rickets.

*Medicinal Treatment.*—In a disease which tends so uniformly to recovery when causal conditions are removed, it is difficult to estimate, by clinical observations, the real value of medicinal treatment. Arsenic and iron are valuable in the treatment of rickets, the special indication for their use being the presence of marked anæmia. Profuse sweating may be relieved by small doses of atropine, i. e., gr.  $\frac{1}{8}$ , three or four times a day, to a child of six months. The special remedies most used are cod-liver oil, phosphorus, and preparations of calcium.

The various preparations of calcium, the phosphate, lactophosphate, and hypophosphite, have long been employed with the belief that they could supply lime to the tissues. It is now practically certain that they do not do so, although at times, they may be useful as tonics in this condition. The two important remedies for rickets are cod-liver oil and phosphorus. No remedy for rickets has held its place so long as has cod-liver oil. Phosphorus, popularised in the treatment of this disease by Kassowitz, has also some value; its most striking results are seen in the early cases and when nervous symptoms are marked. The best results are obtained by a combination of these two remedies. The official oil of phosphorus is used in combination with cod-liver oil, gr.  $\frac{1}{16}$  to  $\frac{1}{8}$  is given three times a day with one-half drachm to one drachm of the oil. Striking confirmation of the clinical observations regarding the value of this combination is furnished by the metabolism experiments of Schabed who found the percentage of calcium retention enormously increased by the use of cod-liver oil and phosphorus.

*Treatment of the Rachitic Deformities.*—The deformities of the chest are less amenable to treatment than most of the others. After the third year something can be done by gymnastics to develop the chest muscles and to increase the pulmonary expansion. The employment of the pneumatic cabinet, in which it is sought to overcome these deformities by the use of rarefied air, has never been given the trial which it deserves. From the very meagre reports published, this appears to be of considerable value.

The deformity of the spine (kyphosis) may usually be overcome by postural treatment. The patient should lie upon a hard bed; no pillow should be allowed under the head, but in severe cases one should be placed beneath the back, so that the head and buttocks are slightly lower than the lumbar spine. While sitting, the shoulders should be kept back and the trunk supported. For a few minutes each day the child should be placed upon the face, and the deformity overcome by raising the but-

tocks while pressure is made upon the spine. In severe cases, an apparatus for giving spinal support, either by a steel brace or a plaster-of-Paris jacket, may be worn a few hours each day when the child is sitting up. Other means should be employed, especially friction and massage, to develop the spinal muscles.

In very many cases slight deformities of the extremities are outgrown when the general treatment can be properly carried out. Where these exist, the physician should take the curve of the legs by seating the child upon a flat surface and tracing their outline with a pencil held perpendicularly. A fresh tracing should be taken once a month. If the deformity is not very great and no increase takes place, it is safe to continue with general treatment only. If the deformity is marked or if it increases in spite of the constitutional treatment, braces should be applied. Something may be done toward straightening the bones by intelligent manipulation. Walking should be discouraged until the bones are quite firm. Friction of the extremities and massage will do very much to increase muscular development. The habit of sitting cross-legged—a very common one in rachitic children—should be prevented, and in fact any other habitual posture, on account of the danger of increasing certain deformities. But little is to be expected from the use of apparatus for the correction of rachitic deformities after the child is two and a half years old; since at this time, and often even at two years, the bones are so firm that no amount of pressure from a steel brace will have any effect.

Without going fully into the question of the surgical treatment of rachitic deformities, for which the reader is referred to text-books of general and orthopædic surgery, I will only state that osteotomy seems to me to offer decided advantages over the other means of treating severe deformities. The best results in osteotomy are obtained when the operation is delayed until the fourth or fifth year, by which time the bones are sufficiently firm and solid. Operations in the second year are generally unsatisfactory, and those in the third year often so, because of the bending of the bones which takes place subsequently. The deformities which require operation are bow-legs and knock-knees, less frequently the curvatures of the femur of the bones of the forearm.

### SECTION III.

#### DISEASES OF THE DIGESTIVE SYSTEM.

##### CHAPTER I.

##### *DISEASES OF THE LIPS, TONGUE, AND MOUTH.*

##### MALFORMATIONS.

**Harelip.**—This is one of the most frequent congenital deformities. It is caused by an incomplete fusion of the central process with one or both of the lateral processes from which the upper half of the face is developed. This deformity may be single or double; the fissure is never in the median line, but usually just beneath the centre of the nostril. There may be simply a slight indentation in the lip, or the fissure may extend to the nostril. Both single and double harelip—more frequently the latter—may be complicated by fissure of the palate. Double harelip is usually accompanied by a fissure between the intermaxillary and the superior maxillary bone of each side.

**Cleft Palate.**—This is second in frequency to harelip. It may involve the soft palate only, or the fissure may extend into the hard palate, producing a wide gap in the roof of the mouth. The most frequent form is that in which only the soft palate is affected.

For the surgical treatment of both these deformities the reader is referred to text-books upon surgery. As to the time of operation, in cases of harelip with a vigorous child of eight or nine pounds, operation in the early days of life is to be preferred. With a small and delicate infant it is best to wait until it is well started in its growth—usually the second month—and in cleft palate during the second year. The medical treatment of these cases consists in the care of the mouth and in the nutrition of the patient. The mouth in all cases must be kept scrupulously clean, but the greatest care is necessary not to injure the epithelium. A camel's-hair brush and plain lukewarm water, or a weak alkaline solution, are to be recommended. Both these deformities are exceedingly likely to be complicated by thrush. This is a serious menace to the success of any operation, and even to the life of the patient. The nutrition is always a matter of much difficulty, and a very large number of these cases die of inanition or marasmus. In cases of harelip, if the

fissure is so great as to interfere with nursing, the child may be fed with a spoon or a medicine dropper until the operation can be done. In cleft palate there may be attached to the rubber nipple of the nursing bottle a flap of thin sheet-rubber in such a way that it closes the fissure in the mouth when once the nipple is in place. This flap should be shaped like a leaf, one extremity being sewed to the neck of the rubber nipple and the other end left free. In many cases, both before and immediately after operation, gavage may be resorted to with the greatest benefit and with very little inconvenience.

**Congenital Hypertrophy of the Tongue.**—This is usually due to disease of the lymphatics, and is to be regarded as a lymphangioma. In a few cases hypertrophy of the muscular fibres has been present. The tongue may reach an enormous size, so that it is impossible for it to be contained within the cavity of the mouth, and it may thus interfere with nursing, deglutition, and even with respiration. The treatment is surgical. Cases like the above are to be distinguished from those of enlargement of the tongue seen in sporadic cretinism. In this disease the tongue is considerably enlarged and may protrude slightly from the mouth, but it is rarely, if ever, large enough to cause other symptoms. It diminishes notably under treatment with thyroid extract.

**Bifid Tongue.**—These cases are extremely rare. Brothers has reported to the New York Pathological Society a case of cleft tongue in a child of one month. There was, in addition, a fissure of the soft palate.

**Tongue-tie.**—This deformity is due to such a shortening of the frenum that it is impossible to protrude the tongue to a normal extent. It differs considerably in degree in different cases. In some, the tongue can not be protruded beyond the gums. Tongue-tie may interfere with articulation, and even with sucking. The treatment consists in liberating the tongue by dividing the frenum with scissors and completing the operation with the finger nail. This should be done in every case unless the child is a bleeder. In many cases the mother may think the tongue tied when the frenum is of normal length.

**Bifid Uvula.**—This is not very uncommon. It usually occurs in connection with cleft palate, but is occasionally seen when there is no other deformity present. It may be complete or partial, and it does not of itself require treatment.

#### DISEASES OF THE LIPS.

**Herpes.**—Herpes labialis is an exceedingly common affection in children, occurring in acute febrile diseases, particularly pneumonia, and sometimes alone. It is the familiar "fever sore" or "cold sore" of domestic medicine. The appearance is similar to herpes in other parts of the body. There is first a group of vesicles, then rupture and the formation of crusts. It is often quite difficult to cure on account of the

disposition of children to pick the lip with the fingers. Although it heals without treatment, recovery is facilitated by the use of some antiseptic lotion, such as dilute boric acid, followed by a dusting powder of zinc oxide and boric acid. This treatment is generally more successful than the use of ointments. Young children should wear mittens or elbow splints at night, to prevent picking at the crusts.

**Eczema of the Lip.**—This is an exceedingly common condition, and a very troublesome one. The vermilion border is dry and rough, and prone to deep cracks or fissures. These are usually seen at the angles of the mouth or in the median line. When severe they are exceedingly painful, bleed freely, and are the cause of very great discomfort, especially in the cold season. The lips should be covered at night by a simple ointment, and this should be used as much as possible during the day. Where deep fissures form, they should be touched with burnt alum, or with the solid stick of nitrate of silver. Syphilitic fissures are considered with the symptoms of that disease.

**Perlèche** (French, *perlécher* = *to lick*).—This name was first given by Lemaistre, in 1886, to a form of ulceration occurring usually at the angle of the mouth. It begins in most cases as a small fissure, which, by constant licking and irritation, to which there is usually added infection, may produce an intractable ulcer of considerable size. It often resembles the mucous patch of hereditary syphilis. The ulcer is of a grayish colour, is quite painful, and is associated with considerable swelling of the lip. It lasts from two to four weeks. The treatment is the same as in simple fissure—viz., the use of burnt alum or nitrate of silver, and covering the part with bismuth or oxide of zinc.

#### DISEASES OF THE TONGUE.

**Epithelial Desquamation.**—This is a disease of the lingual epithelium, which is characterised by the appearance upon the dorsum or margin of the tongue, of circular, elliptical, or crescentic red patches, with gray margins which are slightly elevated. The gray margins are apparently due to thickening of the epithelial layer and the red areas to desquamation of the epithelium. It is sometimes improperly called psoriasis of the tongue. It is quite a common condition, and is probably congenital.

As usually seen, there exist upon the tongue from two to half a dozen of these red patches surrounded by a gray border, which is about one-twelfth of an inch wide, and slightly elevated. The outline of the patch is nearly always crescentic (see Fig. 44). From day to day the configuration of the patches changes; the gray lines advance across the tongue from side to side, or from base to tip, disappearing as they reach the border or the extremity. They are followed by the red patches,

and as the old ones fade away new ones form and run the same course. The red patches are of a bright colour nearest the border, gradually shading off into the normal colour of the tongue. Only the epithelium is involved, the deeper structures being unaffected. The duration of the disease is indefinite; it usually lasts for years. Guinon reports several cases which recovered during an intercurrent attack of measles or scarlet fever.

The cause is unknown. The condition occurs rather more frequently in females than in males, and Gubler has reported an instance of several members of the same family being affected. The condition has been thought to depend upon nearly every disease of childhood. It is not accompanied by pain, salivation, or by other symptoms of stomatitis, and is of little practical importance. Its symptoms are so characteristic that it can hardly be mistaken for any other condition. Treatment is unnecessary.

Two other forms of epithelial desquamation have been observed, both much more rare than that described. In one of these the red denuded portion occupies the margin of the tongue, while the centre is gray or white; the irregular wavy outline which separates the two suggests strongly an outline map, and the condition is sometimes called the "geographical tongue." This term is frequently employed to designate the common form. In another variety nearly the whole organ may be uniformly red, from loss of the epithelium, there being no borders or patches. Both these varieties are of much shorter duration than the more common form, usually lasting only a few weeks.

**Glossitis.**—Inflammation of the tongue is not very common in children. It is usually of traumatic origin. The injury may be due to biting the tongue in a fall or in an epileptic seizure. Glossitis is sometimes excited by the irritation of a sharp tooth, causing a wound which may be the avenue of infection; or it may result from taking into the mouth irritant or caustic poisons. In a small number of cases no cause can be found. The symptoms are marked swelling of the tongue, so that it may protrude from the mouth; and it may even be so great as to cause severe dyspnoea. There are also profuse salivation, difficulty in swallowing and in articulation, and often considerable local pain. There may be a rise of temperature to 102° or 103° F. The treatment consists in the use of fluid food, which in severe cases may be introduced through the nose by means of a catheter. Ice may be used externally, or, bet-



FIG. 44.—EPITHELIAL DESQUAMATION OF THE TONGUE. (Guinon.)

ter still, pieces of ice may be kept in the mouth continually. If there is obstruction to respiration, and in all severe cases, scarification should be done on the dorsum of the tongue along the side of the raphé.

The acute swelling of the tongue and lips occurring in some cases of urticaria may be mentioned in this connection. This is a rare condition in children, but it may develop rapidly and to such a degree as to cause alarming symptoms. The treatment consists in the use of ice locally, free purgation by salines, and, in extreme cases, needle punctures to relieve the œdema.

**Tongue-swallowing.**—This term is used to describe a rare condition seen in infants, in which the tongue is turned backward into the pharynx, so as to obstruct respiration. It may be drawn quite into the œsophagus. Several marked cases have been collected by Hennig.<sup>1</sup> While most frequently occurring with paroxysms of pertussis, tongue-swallowing has been seen in other diseases. This should not be forgotten as one of the explanations of sudden asphyxia in a young infant. The conditions necessary for its production are a somewhat relaxed organ or a long frenum. In none of the fatal cases reported, however, had the frenum been divided. In some weak infants, falling back of the tongue, so that its base partly covers the epiglottis, produces asphyxia, precisely as it occurs in adult life under full anæsthesia. The recognition of the condition is a very easy one, and its treatment is to relieve the obstruction by drawing the tongue forward by the finger or forceps.

**Ulcer of the Frenum.**—The friction against the sharp edges of the lower central incisors frequently causes an ulcer of the frenum in infants. I have never seen it in older children. It usually occurs in pertussis, but is seen in other conditions. In some it appears to be produced by friction of the teeth during nursing from the breast or bottle. It is more often seen in children who are delicate or cachectic than in those who are healthy and well nourished. The ulcer may be confined to the frenum, or it may extend quite deeply into the tongue. It is usually about one-fourth of an inch in diameter, and of a yellowish-gray colour. When not readily cured by touching with alum or nitrate of silver, the child may be fed by gavage for several days, or the teeth may be covered by a bit of absorbent cotton.

#### DENTAL CARIES.

Although the teeth do not strictly belong to the province of the physician they have an important influence upon the general health. The pernicious effects of dental caries have only recently been appreciated. Routine examinations of public-school children, made in various cities, have shown that fully 80 per cent have extensive dental caries. Among

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<sup>1</sup> Jahrbuch für Kinderheilkunde, xi, 299.

the inmates of institutions the proportion is fully as great as this, possibly greater, unless, as in a few modern institutions, special attention is given to this subject.

Among the causes of dental caries the most important without doubt is want of cleanliness—the almost entire neglect of the toothbrush among the children of the poor. This leads to decomposition of food and secretions, acid fermentation, erosions of the enamel, etc. But not all caries of the teeth can be ascribed to this cause. Diet has certainly much to do with it. It is my own belief that the opinion commonly held, that excessive indulgence in sweets is responsible for dental caries, is well founded. Malnutrition and improper food, especially in early childhood, certainly affect the teeth. In some children a congenitally defective enamel is present. Hereditary syphilis is also a cause, and in children with congenital mental defects the teeth are prone to early decay.

The symptoms are both local and general. Locally, as a result of decomposition and infection, there are present foul breath, gingivitis, alveolar abscess, ulcerative stomatitis, toothache, etc. The lymph nodes in the neighbourhood frequently become enlarged and sometimes tuberculous. The tuberculosis of the submaxillary and submental lymph nodes is nearly always the result of infection through the teeth or the gums. Whether the cervical lymph nodes are infected in the same way is very doubtful. The general symptoms result in part from improper mastication of food and in part from sepsis from the local condition. Thus we may have attacks of indigestion, failing nutrition, loss of appetite, and anæmia. From the local irritation various nervous symptoms may arise. The most common are habit spasm, facial chorea, headaches, and according to some writers even epileptiform convulsions. The presence of carious teeth is a menace to the general health. They certainly predispose to local tuberculosis. Many persons assume that if the teeth affected belong to the first set, it matters little. However, the permanent teeth are often injured by extensive decay of the deciduous set. The treatment of this condition belongs to the dentist. But the physician should appreciate the importance of the subject and urge parents and others in charge of children to give proper attention to cleanliness and to see that carious teeth of the first set are either filled or removed.

#### ALVEOLAR ABSCESS.

This is common in children, especially among the class of hospital and dispensary patients, in whom little or no attention is given to the care of the teeth. It causes severe pain and acute swelling, which may be limited to the gum, or it may involve to a considerable extent the periosteum of the jaw and even cause swelling of the whole side of the face.



If there is retention of pus, there may be quite severe constitutional symptoms, such as a chill and high temperature; but in most of the cases these are wanting. The abscess usually opens spontaneously into the mouth, but it may open externally if the molar teeth are the ones affected. It may even lead to necrosis of the jaw. If its site is the upper jaw, the pus may find its way into the nasal cavity or into the maxillary sinus.

The treatment is, in the first place, prophylactic. This requires attention to the teeth to prevent decay, and the removal of old carious fangs, which are a constant menace to the health of the child. The free use of the toothbrush and some antiseptic mouth-wash will, in the great majority of cases, prevent the occurrence of this disease. It is important that the abscess be opened early and free drainage secured. If there is a carious tooth it should be drawn.

#### DIFFICULT DENTITION.

The place of dentition as an etiological factor in the diseases of infancy is one which has given rise to much discussion. From a very early period the view has descended, that a large number of the diseases occurring between the ages of six months and two years are due to difficult dentition. The list of such diseases is a long one, but year by year it has been shortened as one after another has been shown to depend upon other causes, dentition being only a coincidence.

At the present time many good observers deny that dentition is ever a cause of symptoms in children; some even going so far as to say that the growth of the teeth causes no more symptoms than the growth of the hair. Without doubt the usual mistake made in practice is to overlook disease of the brain, ears, lungs, stomach, and intestines, because of the firm belief that the child was "only teething." The physician who starts out with the idea that in infancy dentition may produce all symptoms usually gets no further than this in his etiological investigations. Although no doubt the importance of dentition as an etiological factor in disease has been in the past greatly exaggerated, the careful and candid observer must admit that, particularly in delicate, highly nervous children, dentition may produce many reflex symptoms, some even of quite an alarming character.

Speaking from general impressions not from statistics, I should say that in my experience fully one-half of the healthy children cut their teeth without any visible symptoms, local or general; in the remainder some disturbance is usually seen, and though in most cases it is slight and of short duration, it may last for several days or even a week. The symptoms most commonly seen are disturbed sleep, or wakefulness at night and fretfulness by day, so that children often sleep only one-half

the usual time. There is loss of appetite, and much less food than usual is taken. There is often, but not always, an increase in the salivary secretion, a slight amount of catarrhal stomatitis, and a constant disposition on the part of the child to put the fingers into the mouth. The bowels are often constipated or there may be slight diarrhoea. The thermometer may show a slight elevation of temperature to 100° or 101.5° F. The weight often remains stationary for a week or two, and there may even be a loss of a few ounces. The duration of these symptoms in most cases is but a few days, and they require no special treatment. If the food is forced beyond the child's inclination, attacks of indigestion with vomiting and diarrhoea are easily excited.

Symptoms more severe than the above, are rare in healthy children, but are not infrequent in those who are delicate or rachitic. In such susceptible children, even so slight a thing as dentition may be an exciting cause of quite serious disturbances. Often there is some other factor in the case, such as bad feeding or feeble digestion. In delicate or rachitic children there may be seen the symptoms already mentioned as occurring in healthy infants, but in greater severity; and in addition there may be severe attacks of acute indigestion. Occasionally there is an elevation of temperature to 102° or 103° F., lasting usually only two or three days, and accompanied by no symptoms except almost complete anorexia. Convulsions which could fairly be attributed to dentition I have seen but two or three times; they are more apt to occur in rachitic and highly nervous children. In cases of eczema the symptoms often undergo a distinct exacerbation with the eruption of each group of teeth. As regards almost all the other diseased conditions which are commonly attributed to dentition, I believe that it is a delusion to ascribe them to this cause.

The physician should watch a child carefully, and examine it frequently, to be sure that he is not overlooking some serious local or constitutional disease before he allows himself to make the diagnosis of difficult dentition. Probably in ninety-five per cent of the cases in which symptoms are present, they are due to some cause other than dentition. When, however, symptoms such as any of those mentioned disappear immediately when the teeth come through, and when we see them repeated four or five times in the same child with the eruption of each group of teeth, and accompanied by red and swollen gums, I think we can not escape the conclusion that dentition is a factor in their production, though perhaps not the only one.

In the treatment of this condition drugs occupy but a small place. It should be remembered that infants are at this time in a peculiarly susceptible condition as regards the digestive tract, and attacks of indigestion, and even severe diarrhoea, are readily excited from slight causes, especially from overfeeding. Special care should be exercised in this

respect. The strength of the food should be reduced, as well as the amount given. A poor appetite indicates a feeble digestion, which should not be overtaxed. As attacks of bronchitis and acute nasal catarrh are readily induced, even slight exposure should be guarded against. The nervous symptoms, when severe, may be relieved by the use of moderate doses of the bromides or by phenacetine, better than by opiates. All soothing syrups should be discountenanced. All the various devices for making dentition easy are a delusion. In a small number of cases lancing the gums is of value. I have myself seen in a few rare instances marked and undoubted relief given by it. This is likely to be the case where the gums are tense, swollen, and very red, with the teeth just beneath the mucous membrane. To press a tooth through the gum by simply rubbing gently with the finger covered with sterile gauze is frequently more effective than an incision. It is seldom, however, that the relief expected is seen from any of these measures.

#### CATARRHAL STOMATITIS.

This is characterised by redness and swelling of the mucous membrane, and by increased secretion of the salivary and the muciparous glands of the mouth. It usually involves a large part of the mucous membrane.

**Etiology.**—Catarrhal stomatitis may result from traumatism. This injury may be mechanical, or due to heat or any irritant accidentally taken into the mouth. It frequently occurs at the time of the eruption of a tooth. It complicates measles, scarlet fever, diphtheria, influenza, and many other infectious diseases. In these cases, and in many others, the disease is probably due to direct infection.

**Lesions.**—The lesions are essentially the same as in catarrhal inflammations of other mucous membranes. There are congestion with desquamation of epithelial cells, and sometimes the formation of superficial ulcers. The process may be a very superficial one, or it may extend to the submucous tissue.

**Symptoms.**—The mucous membrane is intensely injected, all the capillaries are dilated, and small hæmorrhages easily excited. The mucous membrane is swollen, this being most apparent over the gums or about the teeth. There may be some swelling of the lips. The mouth seems hot, and the local temperature is certainly increased. There is considerable pain, as shown by fretfulness, but particularly by the disinclination to take food: infants, though evidently hungry, either refusing the breast or bottle altogether, or dropping it after a few moments. The increase in secretion is sometimes marked, so that the saliva pours from the mouth, irritating the lips and face and drenching the clothing. In other cases the saliva is swallowed. On close inspection there may be

seen swelling of the muciparous follicles, and even the formation of tiny cysts from the accumulation of secretion within them. The tongue is usually coated, the edges reddened, and the papillæ prominent. In febrile diseases, such as typhoid, etc., we may get an accumulation of dead epithelium with the formation of cracks and fissures of the tongue, and the lips may present a similar condition. The neighbouring lymphatic glands are slightly enlarged and tender. The constitutional symptoms accompanying simple stomatitis are not severe, but some disturbance is almost always present. There may be derangement of digestion with vomiting, and even a mild attack of diarrhœa. In the majority of cases the disease runs a short course, recovery taking place in a few days when the primary cause is removed. In very delicate children it may be prolonged, and from the interference with nutrition may even lead to serious consequences.

**Treatment.**—The mouth and teeth should be kept clean. Food is more acceptable if given cold. In very severe cases, where food is refused, gavage may be resorted to three or four times daily. In all cases children may be given ice to suck. This is refreshing, both on account of the cold and from the relief to the thirst. The mouth should be kept clean with a solution of boric acid, ten grains to the ounce, or an alkaline solution, such as Dobell's, diluted with an equal amount of cold boiled water; or plain water may be used. In the severe forms, where there is much swelling and slight catarrhal ulceration, astringents are required. In my experience alum is the best; this may be applied in the form of the powdered burnt alum mixed with an equal amount of bismuth, or in solution, ten grains to the ounce, with a swab or brush. Where ulcers are slow in healing and very painful, the powdered burnt alum or the solid stick of nitrate of silver may be applied directly.

### HERPETIC STOMATITIS.

*(Aphthous, Vesicular, or Follicular Stomatitis.)*

In this form of stomatitis we have the appearance first of small yellowish-white isolated spots, and subsequently the formation of superficial ulcers. These ulcers are first discrete, but may coalesce and form others of considerable size. It is a self-limited disease, usually running its course in from five days to two weeks.

**Etiology.**—Very little is as yet positively known regarding the cause of herpetic stomatitis. It is not common in the first year, but after that is very frequently seen throughout childhood. It occurs in the strong as well as in the delicate. It is often associated with some disturbance of the stomach, and occasionally with dentition. I have adopted the term herpetic because the condition is analogous to herpes of the lips and face, the difference in appearance being due chiefly to location. It is

apparently caused by something which acts upon terminal nerve filaments.

**Lesions.**—The generally accepted opinion is that there is first a vesicle, followed by a death of epithelial cells covering it, and then a superficial ulcer. The white appearance is due to the fact that the ulcers, being on a mucous membrane, are always moist. These ulcers may extend superficially, but never deeply; they heal quickly with the formation of new epithelial cells, leaving no cicatrices. Herpetic stomatitis is always associated with more or less catarrhal inflammation.

**Symptoms.**—The disease is characterised by local and general symptoms. The latter are quite indefinite—general indisposition, loss of appetite, and slight fever. The local symptoms consist in the development of small, shallow, circular ulcers, usually coming in successive crops. While most frequent at the border of the tongue and the inside of the lips, they may be found upon any part of the mucous membrane of the mouth or the pharynx. There may be only half a dozen present, or the mouth may be filled with them. They are first of a yellowish colour, and on an average about one-eighth of an inch in diameter. By the coalescence of several smaller ones there may form patches of considerable size, sometimes nearly covering the lips. The older ulcers are apt to have a dirty grayish colour, and in places may look not unlike a diphtheritic membrane. The smaller ones are surrounded by a red areola, and when healing the margin is of a bright red colour. Their appearance is often more like that of an exudation upon the mucous membrane than an excavation into it. The other symptoms are much the same as those of catarrhal stomatitis, but usually of greater severity. The pain is particularly intense, it being often difficult to induce children to take anything in the form of food. The tongue is frequently coated, but there is never the foul breath of ulcerative stomatitis. The duration of the disease is from one to two weeks, and, if the child is in good condition, complete recovery takes place even without any special treatment. In badly nourished children the disease may last for two or three weeks; relapses may occur, and the condition may interfere very seriously with the child's nutrition.

**Treatment.**—This is the same as in catarrhal stomatitis, with the addition that to each one of the ulcers finely powdered burnt alum should be applied with a camel's-hair brush. If this is not effective, the solid stick of nitrate of silver may be used. The ulcers will usually yield rapidly to this treatment. In my experience, drugs given with the purpose of affecting the lesion in the mouth have been without benefit.

#### ULCERATIVE STOMATITIS.

Ulcerative stomatitis is believed to occur only when teeth are present. It is characterised by an ulcerative process, beginning at the junc-

tion of the teeth and the gum, and extending along the teeth; it occasionally involves other parts of the mouth, but never spreads beyond the buccal cavity.

**Etiology.**—A form of ulcerative stomatitis is produced by certain metallic poisons, especially mercury, lead, and phosphorus; but all these are now rare. Ulcerative stomatitis also occurs in scurvy; and it seems probable that an allied disturbance of nutrition, with spongy, swollen gums, precedes some other forms of ulcerative stomatitis. Bad surroundings and improper food act as predisposing causes; for the disease is quite common in institutions for children and in hospital and dispensary patients, although rare in private practice. Local causes of importance are want of cleanliness of the mouth and teeth and the presence of carious teeth. Conditions which produce a lowered vitality of the gums act as a predisposing cause, and infection as an exciting cause of the disease. The constant clinical features of ulcerative stomatitis and the occasional occurrence of epidemics indicate a specific cause which is probably the same as that of ulcero-membranous tonsillitis. The two conditions often exist at the same time. From the investigations of Vincent, Bernheim, Plaut and others it seems probable that noma is also produced by the same organism but represents a more virulent infection.

**Lesions.**—The disease may begin at any part of the mouth, but most frequently upon the outer surface of the gum along the lower incisor teeth. From this point it extends behind the teeth, and from the incisors to the canines and molars, usually of one side only; but it may involve the entire gum of both jaws. From the gums the process may spread to the lips, affecting the fold of mucous membrane between the gum and the lip, and also to the inner surface of the cheek, especially opposite the molar teeth, where large ulcers often form. In neglected cases the disease may extend into the alveolar sockets, the teeth loosening and falling out. The periosteum of the alveolar process may be involved, and even superficial necrosis of the jaw may occur, as has happened in several cases that came under my observation. These severe forms are met with in institutions chiefly and then generally follow measles or scarlet fever.

Ulcers similar in appearance may also be present in other parts of the mouth—i. e., on the soft palate or the tonsils, sometimes even when the gums are not involved.

**Symptoms.**—The first things noticed are the very offensive breath and the profuse salivation. It is usually for one of these symptoms that the patient is brought for treatment. On inspection of the mouth, there are seen in the mild cases, swollen, spongy gums of a deep-red or purplish colour, which bleed at the slightest touch. There is a line of ulceration, usually along the incisor teeth, most marked in front, which may extend to any or to all of the teeth; sometimes it affects only the gum

along the molar teeth, the incisors escaping. At the junction of the teeth and gum is seen a dirty, yellowish deposit, on the removal of which free bleeding takes place. The diseased parts are very painful, and the child cries and resists any attempt at examination. In the more severe cases and in those of longer duration the teeth are loosened, sometimes being so loose that they can be picked from the gum. There may be necrosis of the jaw, and even a loose sequestrum may be found. In these cases the ulceration along the gums is deeper, and there may be ulcers in the cheek opposite the molar teeth, or inside the lip. The swelling may be so great that the teeth are almost covered; this is seen particularly in the scorbutic form. The saliva pours from the mouth, adding greatly to the discomfort of the patient. Beneath the jaw are felt the large, swollen lymphatic glands, which are painful and tender to the touch, but show no tendency to suppurate. The tongue is somewhat swollen, and shows at the edges the imprint of the teeth; it has a thick, dirty coating.

The disease is attended by little or no fever or other constitutional symptoms. The general condition of these patients is often poor, and there may be quite a marked cachexia. Other forms of stomatitis may be associated, and it should not be forgotten that the gangrenous form may follow.

When not recognised or not properly treated, ulcerative stomatitis may last for months. When properly treated it tends in all recent cases to recovery, usually in from five to ten days. No deformity of the mouth is left, the only untoward results being shrinking of the gum, sometimes loss of some of the incisor teeth, and more rarely a superficial necrosis of the alveolar process. All these are quite uncommon. Ulcerative stomatitis can hardly be confounded with any other form, and not only should a diagnosis of the lesion be made, but the condition upon which it depends should, if possible, be discovered; scorbutus, particularly, should not be overlooked.

**Treatment.**—The first thing to be done is to remove the cause. When dependent upon metallic poisoning the source should be discovered. Scorbutic cases should have the usual anti-scorbutic diet. Cleanliness of the mouth is of great importance, and this may best be accomplished by the use of peroxide of hydrogen diluted with from one to four parts of water. It should be followed by thorough rinsing with plain water, and repeated several times a day. In other cases a solution of alum, five grains to the ounce, or a mouth-wash of chlorate of potash, three grains to the ounce, may be employed. The only objection to the last mentioned is the pain which it usually produces.

The specific remedy for ulcerative stomatitis is chlorate of potash. The best method of administration is to give two grains, or one-half teaspoonful of a saturated solution, largely diluted, every hour during the

day for the first twenty-four hours and subsequently every two hours; when improvement occurs the dose may be still further reduced. Marked benefit is usually seen in one or two days even in cases that have lasted for several weeks. If the case does not yield readily to this treatment there is probably disease at the roots of the teeth, and when loose these should be removed, and the jaw examined to see if there is necrosis. Occasionally when there is no disposition to heal, the shreds of necrotic tissue should be carefully removed, and burnt alum or nitrate of silver applied.

The constitutional and dietetic treatment in all these cases should be the same as that employed in scurvy—i. e., plenty of fruit, fresh vegetables, and sometimes the internal administration of mineral acids, especially aromatic sulphuric acid. Iron is indicated in most of the cases.

**Ulceration of the Hard Palate.**—This is usually seen in the first few weeks of life, but may occur in any child suffering from marasmus. The primary cause may be the injury inflicted in cleansing the mouth. In other cases it is due to the friction of the rubber nipple, or some other object which the child is allowed to suck. In still others it is apparently produced by the habit of tongue-sucking frequently observed in these young infants. The appearances are quite characteristic: there is found, rather far back upon the hard palate, usually in the middle line, a superficial ulcer, from a fourth to a half inch in diameter. There are no signs of acute inflammation. Thrush may coexist, but it has no relation to the production of the disease. Spontaneous recovery usually occurs in from one to three weeks, provided the cause can be removed. In children suffering from marasmus these ulcers are very intractable, and in many instances their cure is practically impossible. It is therefore especially important to prevent, if possible, their formation by care in cleansing the mouth, and in avoiding the other causes referred to. When ulcers have appeared they should be treated as in cases of herpetic stomatitis.

#### THRUSH.

(*Sprue; German, Soor; French, Muguet.*)

Thrush is a parasitic form of stomatitis characterised by the appearance upon the mucous membrane, usually of the tongue or of the cheeks, of small white flakes or larger patches. It is common in infants of the first two or three months, and in all the protracted exhausting diseases of early life.

**Etiology.**—The exact class to which the vegetable parasite which produces thrush belongs has not yet been definitely settled. Robin's opinion was long accepted that it was the *oïdium albicans*; the view of Grawitz, that it is the *saccharomyces albicans*, is now more generally adopted. If a little of the exudate from the mouth is placed upon a



slide and a drop of liquor potassæ added, the structure of the fungus is readily seen. With the low power of the microscope there can be made out fine threads (the mycelium) and small oval bodies (the spores). With a high power the threads can be seen to be made up of a number

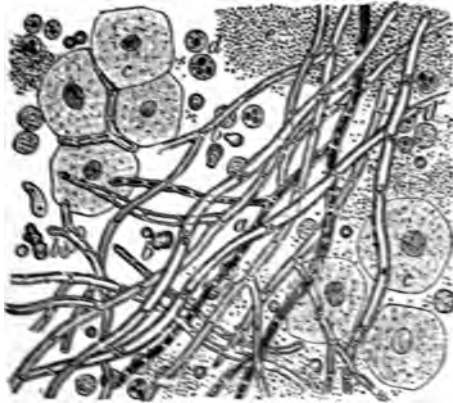


FIG. 45.—THRUSH FUNGUS (highly magnified).  
a. mycelium; b. spores; c. epithelial cells from the mouth; d. leucocytes; e. detritus. (v. Jaksch.)

of shorter rods, at the ends of which the spore formation takes place (Fig. 45). The mycelium is produced from the spores. The spores of this fungus are of very common occurrence in the atmosphere. It is difficult or impossible for thrush to develop upon a healthy mucous membrane. Its growth is favoured by slight abrasions, such as are often produced by rough methods of cleansing the mouth; also by catarrhal stomatitis, a scanty salivary secretion and want of cleanliness.

The nature of the process which it produces is in all probability a sugar fermentation, the acid reaction of the mouth being the result of the growth rather than its cause. Infection may come from another patient by means of a rubber nipple or a cloth which has been used for the infected mouth, from the nipple of the nurse, or directly from the air. Its production is favoured by a scanty secretion of saliva, hence it is frequent in the first two or three months of life; also by an altered secretion such as is seen in protracted wasting diseases, enterocolitis, marasmus, typhoid, tuberculosis, etc. It is very common in infants suffering from hardlip or any other deformity of the mouth. The disease is frequently seen in foundling asylums, in all places where many young infants are crowded together, and where cleanliness of mouths, bottles, etc., is neglected.

**Lesions.**—The spores lodge between the epithelial cells and gradually separate the different layers. This occurs before the formation of the white pellicle. Later the disease spreads on the surface of the mucous membrane, and also penetrates the deeper structures. It may invade the blood vessels and cause thrombosis or even be carried to distant parts. Although the *saccharomyces albicans* is commonly found upon fat epithelium, its growth is not confined to it. It usually begins at many distinct points upon the mucous membrane, and gradually spreads until coalescence takes place, a continuous membrane may be thus formed. No pus is produced by the process.

The usual seat is the margin of the tongue, the inside of the lips and cheeks, and the hard palate, but not infrequently it involves the pillars of the fauces, and the pharynx. Further extension in the digestive tract than this is rare, although the stomach, and even the intestines, may be invaded. I have seen it but once or twice in the œsophagus and never in the stomach, and I know of but two reported cases in this country in which thrush has been found there. Cases involving the œsophagus and the stomach appear from reports to be much more common in Europe. In several cases in the Babies' Hospital the *saccharomyces albicans* has been found in the lungs of infants suffering from broncho-pneumonia. There are several reported cases of general blood infection from this organism.

**Symptoms.**—The essential symptoms of thrush are the appearance upon the mucous membrane of the mouth—usually beginning upon the tongue or the inner surface of the cheek—of small white flakes which resemble deposits of coagulated milk, but which differ from them in the fact that they can not be wiped off. If forcibly removed, they usually leave a number of bleeding points. There may be only a few scattered patches, or the mouth and pharynx may be covered. The mouth is generally dry, the tongue coated; food may be refused on account of pain, and there may be some difficulty in swallowing. The other symptoms depend upon the conditions with which the thrush is associated.

**Diagnosis.**—This is rarely difficult. The deposit may be mistaken for coagulated milk, but is distinguished by the features just mentioned. When existing upon the pharynx and fauces it has been confounded with diphtheria, although this mistake can hardly be made if all the facts of the case are taken into consideration—the age of the patient, the involvement of the lips and tongue, the dry mouth, the absence of glandular enlargement, etc. In any case of doubt the examination of the deposit under the microscope at once reveals its true nature.

**Prognosis.**—Thrush is not in itself a dangerous disease, except in the very rare instances where it may obstruct the œsophagus, and this can hardly occur except in a condition of exhaustion which is necessarily fatal. In a feeble and delicate infant, or in one with harelip or cleft palate, thrush may be a serious complication. With proper treatment most of the cases involving only the mouth are readily cured.

**Treatment.**—Thrush may usually be prevented by due attention to cleanliness of the mouth, rubber nipples, bottles, cloths, etc. In infants with deformities of the mouth in institutions, it frequently develops despite all precautions. All rubber nipples should be kept in a solution of boric acid and the child's mouth should be cleansed several times a day. On no account should a feeding-bottle be passed from one child to another.

In the treatment of the disease the essential things are cleanliness,

and the use of some mild antiseptic mouth-wash. The best routine treatment is to cleanse the mouth carefully after every feeding or nursing with a solution of bicarbonate of soda, and to apply twice a day a one-per-cent solution of formalin. All applications should be carefully made, so as not to injure the epithelium. The best method of cleansing is by a small swab made with a wooden toothpick and absorbent cotton. Applications to be especially avoided are those mixed with honey or any syrup. In hospital cases the disease seems to be prolonged by the irritation of the rubber nipple of the feeding-bottle. In such it has been our practice to feed by gavage for two or three days, as some cases improved much more rapidly when this was done.

#### GONORRHOËAL STOMATITIS.

There has been described by Dohrn and Rosinski a form of stomatitis in the newly born, due to a gonorrhœal infection. This is not likely to take place unless the epithelium has been removed. The infection in all cases occurred from the mother. The lesion consists in the formation of yellowish-white patches upon the tongue or hard palate—regions in which the epithelium is liable to be injured by rough attempts at cleansing the mouth. There may be other evidences of gonorrhœal infection, especially ophthalmia. The diagnosis rests upon the discovery of the gonococcus in the exudate. In all the cases cited the general health was not affected, and recovery followed in the course of a week or ten days.

The treatment consists in thorough cleanliness and in the application of a saturated solution of boric acid, as in thrush.

#### SYPHILITIC STOMATITIS.

The buccal symptoms of hereditary syphilis are important both from a diagnostic and a therapeutic standpoint. The most frequent lesions are fissures, ulcers, and mucous patches. Fissures are found upon the lips, most frequently at the angle of the mouth, and are usually multiple. They may be quite deep and cause frequent hæmorrhages. Mucous patches are superficial ulcers developing from papules which form upon the mucous or muco-cutaneous surfaces. In cases of acquired syphilis in children the primary sore may be seen upon the tongue, the lip, or the tonsil. All these symptoms are more fully considered in the chapter on Syphilis.

#### DIPHTHERITIC STOMATITIS.

In severe cases of diphtheria the membrane is found not only upon the pharynx and tonsils, but it may appear anywhere upon the buccal mucous membrane or the lips. It is questionable whether the diphtheritic process ever begins on the mucous membrane of the mouth, or is

ever limited to this part. In my own experience diphtheritic stomatitis has always been associated with deposits upon the tonsils and pharynx. It is seen only in the severest cases, and in those which, from other conditions present, are almost necessarily fatal. Bearing in mind the above points, it can hardly be mistaken for any other variety of stomatitis, although not infrequently the mistake is made of regarding as diphtheritic, cases of herpetic stomatitis in which the ulcers have coalesced. The treatment, so far as the mouth is concerned, consists in cleanliness by frequent gargling or irrigation with a hot saline solution. Forcible removal of the membrane is not to be advised.

#### GANGRENOUS STOMATITIS—NOMA.

(*Cancrum oris.*)

The term noma is used to designate all forms of spontaneous gangrene occurring in children, which involve mucous membranes or mucocutaneous orifices. The most frequent situation being the mouth, noma and gangrenous stomatitis are often used synonymously. Noma may, however, affect the nose, external auditory canal, vulva, prepuce, or anus. It is a rare disease, and usually terminates fatally.

**Etiology.**—Noma is seldom seen outside of institutions for children, where small epidemics are not uncommon. It is usually secondary to some of the infectious diseases, most frequently following measles, and next to this scarlet fever, typhoid, or whooping-cough. While it may occur at any age, most of the cases are in children under five years, and in those of poor general condition. Noma seldom attacks parts previously healthy. In the mouth it may be preceded by catarrhal, or more often by ulcerative stomatitis; in the auditory canal, by a chronic otitis media. There seems little doubt that the disease is contagious. In 1899 I saw five cases in a single ward, all beginning in the auditory canal, which were apparently produced by the use of the same syringe to clean the ears without proper disinfection. All these children were suffering from whooping-cough at the time.

It is now quite well established that the exciting cause of noma is the same as that of ulcerative stomatitis (q. v.). The pathological process in one case is of a mild type occurring in patients of considerable resistance. In the other it is of a severe or malignant type occurring in patients of feeble resistance as a result of previous acute disease. In the gangrenous tissue pyogenic cocci and putrefactive bacteria are abundant. In the border zone, and extending into the adjacent healthy tissue the specific organisms of the disease are found.

**Lesions.**—The process is one of slowly spreading gangrene. In most of the cases there are thrown out inflammatory products in quite large amount, but there is little or no tendency to limitation of the disease.

This usually advances steadily until death occurs. In a small number of cases a line of demarcation finally forms, and the slough separates, leaving a large area to be partially filled in by granulation and cicatrisation. Other infectious processes are likely to accompany the disease, particularly broncho-pneumonia.

**Symptoms.**—The constitutional symptoms are not usually severe until the local disease has existed for several days. Then those of marked prostration and sepsis develop, sometimes quite rapidly. The temperature is usually elevated to 102° or 103° F., and sometimes to 104° or 105° F. There are dulness, apathy, feeble pulse, muscular relaxation, and very often diarrhoea. Before death the temperature may be sub-normal.

Of the local symptoms, often the first to attract attention is the odour of the breath; sometimes it is the dusky spot on the cheek or lip. On examination of the mouth, there usually is found upon the gum or inside of the cheek a dark, greenish-black necrotic mass, surrounded by tissues which are swollen and oedematous, so that the cheek or lips may be two or three times their normal thickness. Externally the parts are tense and brawny from the swelling, this infiltration always extending for some distance beyond the gangrenous part. As the process extends, the teeth loosen and fall out; there may be necrosis of the alveolar process of the jaw and perforation of one or both cheeks or lower lip. Extensive sloughing of the face may take place, usually upon one side, sometimes upon both, giving the patient a horrible appearance, as shown in Fig. 46. In this patient the process began in the right cheek, subsequently involving the left; perforation occurred in both cheeks, and before death a large part of the face was gangrenous. The odour from a severe case is very offensive, and, in spite of all efforts at disinfection, it may fill the ward or even the house. Pain is rarely severe, and in many cases it is absent. Extensive hæmorrhages are rare.

I have notes of seven cases in which noma affected the ear, being preceded by chronic otitis media in every instance. The disease began in the deeper structures of the canal, the first symptom noticed usually being a nodular swelling just beneath the ear, crowding the lobe upward. Shortly afterward there appeared the dirty brown discharge with a gangrenous odour. Later, the gangrenous circle surrounded the meatus, which gradually extended, until in some cases the whole side of the face and head were involved. A probe could readily be passed into the cranial cavity. All these cases ended fatally.

The usual duration of the disease is from five to ten days. If recovery takes place, there is first seen a line of demarcation; then the slough is thrown off, and granulation and cicatrisation begin, but require a long time, usually leaving an unsightly deformity.

The prognosis is grave, fully three-fourths of the cases proving fatal.

The results depend not only upon the disease itself, but upon the condition of the patient with which it is associated.

Gangrenous stomatitis can hardly be mistaken for any other form of disease occurring in the mouth, and early recognition is of great importance, since only early treatment is likely to be successful.



FIG. 46.—GANGRENOUS STOMATITIS, FOLLOWING MEASLES.  
(From a photograph lent by Dr. Henry Moffat.)

**Treatment.**—Much can be done to prevent the disease by careful attention to all the milder forms of stomatitis, particularly to the ulcerative variety. Frequent and thorough cleansing of the mouth in all acute infectious diseases is a part of the treatment which is too often neglected. This should be a matter of routine in every severe illness in a young child. Recognising the malignant nature of gangrenous stomatitis, its treatment should be radical from the very outset. Of the measures which have been proposed, that which seems to offer the best chance of

arresting the process is excision with cauterisation. This should be done under anæsthesia. In excising, one should go some distance into tissues apparently healthy, for the reason that the process has always advanced farther in the subcutaneous tissues than in the skin. The edges of the wound should then be thoroughly cauterised, best by the Paquelin cautery. Of the other means employed, the use of strong carbolic acid immediately followed by alcohol is probably the best. This is to be used after excising or curetting the necrotic tissue. The mouth should be kept as clean as possible by the use of peroxide of hydrogen. The general treatment should be supporting and stimulating. As the possibility of contagion exists, every case should be isolated.

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## CHAPTER II.

### *DISEASES OF THE PHARYNX.*

#### ACUTE PHARYNGITIS.

ACUTE pharyngitis may exist as a primary disease, or with any of the infectious diseases, particularly scarlet fever, measles, diphtheria, or influenza. Secondary pharyngitis will be considered in connection with these different diseases.

Certain children have a constitutional predisposition to attacks of acute pharyngitis, and contract it upon the slightest provocation. In some of them there is a strongly marked rheumatic diathesis. Attacks of acute pharyngitis often follow exposure. In many cases they are associated with acute disturbances of digestion. All of the above causes probably act by producing local and general conditions favourable to the development of micro-organisms already present in the mouth. The bacteria most frequently associated with severe attacks are the staphylococcus, the pneumococcus, the streptococcus, and less frequently, the bacillus influenzae.

In acute catarrhal pharyngitis the inflammation may involve the entire mucous membrane of the tonsils, fauces, uvula, posterior and lateral pharyngeal walls, or any part of it. It may exist alone, or in connection with a similar inflammation in the rhino-pharynx or in the larynx. In the beginning there is seen an acute redness, usually involving the entire pharynx. This may entirely subside after twenty-four hours, or it may be followed by the usual changes of acute catarrhal inflammation—dryness, swelling, and œdema. Later there is increased secretion of mucus, and finally there may be muco-pus. Occasionally slight hæmorrhages are present.

There is pain at the angle of the jaws, which is increased by swallow-

ing, also a sensation of dryness and roughness in the pharynx, and often an irritating cough. There may be slight swelling of the neighbouring lymphatic glands. The constitutional symptoms in young children are often severe. Not infrequently there is a sudden onset with vomiting, and a rise of temperature to 102° or even 104° F. These symptoms are usually of short duration, frequently less than twenty-four hours, and in two or three days the patient may be entirely well. In other cases the pharyngitis may be accompanied or followed by laryngitis.

Acute primary pharyngitis is to be distinguished from scarlet fever, diphtheria, measles, and influenza. A positive diagnosis from scarlet fever is impossible until a sufficient time has elapsed for the eruption to appear, and the patient should be closely watched for the first sign of this. If scarlet fever is prevalent, a child with the symptoms of severe pharyngitis should at once be isolated while waiting for the diagnosis to be settled. There is commonly less difficulty in excluding measles because of the absence of Koplik's sign on the buccal mucous membrane, and of the accompanying catarrh of the eyes and nose. Catarrhal diphtheria can be excluded only by culture.

The first step in the treatment of acute pharyngitis is to open the bowels freely by means of calomel, castor oil, or magnesia. The child should be kept in bed, and the diet should be fluid, or, in the case of infants, the amount of food should be much reduced. Pieces of ice may be swallowed frequently for the relief of pain and thirst. Internally there may be given two grains of phenacetine every four hours to a child of three years. It is important at the outset to induce free perspiration. The disease is not serious, and the indications are to make the child as comfortable as possible during the short attack. I have seen but little benefit from the use of aconite, although for years I saw it used as a routine treatment.

#### UVULITIS.

Acute inflammation of the uvula, with swelling and œdema, occurs as a part of the lesion in acute pharyngitis. In rare instances the uvula may be the principal or the only seat of inflammation. Huber (New York) has reported two cases, one of which is unique. An infant ten months old was apparently well until two hours before it was seen, when there was noticed a constant irritating cough, accompanied by considerable gagging. Later there could be seen in the mouth a prominent red mass, the enlarged and elongated uvula. It was accompanied by paroxysms of coughing, which interfered both with nursing and deglutition. The general symptoms were quite alarming. The uvula was found to be fully one inch long and half an inch wide, red and œdematous; in other respects the throat was normal. The symptoms were relieved by multiple



needle punctures and the use of ice. In such conditions the greatest relief is often afforded by the application of adrenalin, or its use as a spray or gargle.

#### ELONGATED UVULA.

Probably this is primarily a congenital condition. It is increased by repeated attacks of acute or subacute inflammation. The degree of elongation varies in different cases; in some it may reach an inch in length. Only the mucous membrane is involved in the elongation. The symptoms are those of local irritation, especially a cough upon lying down, and the sensation of a foreign body in the pharynx. In some cases it may be a reflex cause of asthma, or, more frequently, of catarrhal spasm of the larynx. The diagnosis is very easily made by inspecting the throat. The treatment consists in grasping the tip of the uvula with forceps and cutting off the excess with the scissors, or a uvulotome. Care should be taken not to cut off too much of the uvula, or severe hæmorrhage may occur.

#### RETRO-PHARYNGEAL ABSCESS.

Two distinct varieties are seen: (1) The so-called idiopathic abscesses which belong to infancy, and (2) abscesses secondary to caries of the cervical vertebræ.

**Retro-pharyngeal Abscess of Infancy.**—All of the later investigations regarding this disease go to show that primarily it is not a cellulitis, but a suppurative inflammation of the lymph nodes (lymphatic glands) with a surrounding cellulitis. The retro-pharyngeal lymph nodes form a chain on either side of the median line between the pharyngeal and the prevertebral muscles. These nodes are said to undergo atrophy after the third year, and in some cases to disappear entirely. Retro-pharyngeal abscess—or, more properly, retro-pharyngeal lymphadenitis, since the process does not invariably go on to suppuration—is probably never primary, but secondary to infectious catarrhs of the pharynx, and is set up by the entrance of pyogenic bacteria, usually the staphylococcus or streptococcus. Its pathology is the same as the more frequent suppurative inflammation of the external cervical lymph nodes, with which it is sometimes associated. Usually only a single node is involved, but sometimes two or three are affected, and these may be situated upon opposite sides. I have frequently seen retro-pharyngeal lymphadenitis so severe as to give rise to marked local symptoms, although it did not go on to suppuration. Kormann's observations, however, show that swelling of these glands in diseases of the mouth and throat is very much more common than is generally supposed. Similar abscesses from suppurative inflammation of other lymph nodes in the neighbourhood

of the pharynx may occur. I have seen one situated between the epiglottis and the base of the tongue.

*Etiology.*—These cases almost invariably occur in infancy. Fully three-fourths of those that have come under my observation have been in patients under one year. Bokai (Buda-Pesth) reports that of sixty cases observed, forty-two occurred during the first year, eleven during the second year, and only seven at a later period. The primary disease is usually a severe rhino-pharyngitis, or an attack of epidemic influenza, but rarely it occurs as a sequel of scarlet fever or measles. In six hundred and sixty-four cases of scarlet fever, Bokai noted retro-pharyngeal abscess in seven cases. After measles it is even more rare. Retro-pharyngeal abscess usually occurs in winter or spring, on account of the prevalence of the diseases upon which it depends. It is seen in children previously robust, but more often in those who are delicate and who in consequence are prone to severe catarrhal affections.

*Symptoms.*—The early symptoms in most cases are merely those of an ordinary rhino-pharyngeal catarrh. After this has subsided the temperature may remain slightly elevated, often for a week or more, before local symptoms are noticeable. Sometimes, without any distinct history of previous catarrh, there are seen quite high temperature, from 102° to 104° F., loss of flesh, and prostration. A careful examination may be required, and sometimes observation for a day or two, before the explanation of these constitutional symptoms is discovered. In other cases the early constitutional symptoms are so slight as to escape notice, and the local symptoms are the only ones present. Although usually these are not severe, retro-pharyngeal abscess may cause dyspnoea, which in a short time assumes an alarming character. The duration of the inflammatory process before abscess forms is generally five or six days, but it may be several weeks. The temperature is invariably elevated, usually from 100° to 103° F.; occasionally it may be 104° or 105° F., with symptoms of prostration seemingly out of all proportion to the local disease, but which are to be explained by the tender age and feeble resistance of the patient.

The most characteristic local symptoms are the posture, the head being drawn far backward to relieve pressure on the larynx, the noisy respiration with the mouth open, and difficulty in deglutition. Sometimes the first thing to attract notice is a sudden attack of dyspnoea severe enough to cause asphyxia. This is due to the pressure forward of the abscess encroaching upon the larynx. The mouth may be dry, or there may be a copious secretion of pharyngeal mucus. The dyspnoea is in most cases greater on inspiration, and in some it is noticed only then, expiration being normal. The difficulty in swallowing is greater when the tumour is low. The child may find it impossible to swallow, and in consequence may refuse to nurse; or the difficulty in nursing

may depend upon the nasal obstruction. Sometimes there is regurgitation of food through the nose or mouth. The voice is usually nasal. Generally there is no hoarseness, but a peculiar short cry which is quite characteristic. There may be, although rarely, aphonia. Usually there is some swelling to be seen externally, just below the angle of the jaw in front of the sterno-mastoid muscle; exceptionally this may be more prominent than the internal swelling. In one or two cases I have noticed torticollis as an early symptom.

A positive diagnosis is made by an examination of the throat. On inspection there is seen a distinct bulging of the lateral wall of the pharynx, usually a little above the base of the tongue. The swelling may be so great as to crowd the uvula to one side and nearly fill the pharynx. It is rarely if ever in the median line. There is usually redness of the mucous membrane and œdema of the uvula and of the adjacent parts. On digital examination the swelling is made out even better than by inspection. It may be situated so low down as not to be visible at all. In the early stage there may be felt only a localised induration or a somewhat diffuse swelling, but by the time the swelling is large enough to produce marked symptoms, fluctuation can generally be discovered.

*Prognosis.*—When left to itself the abscess may open into the pharynx, the pus being swallowed or expectorated. The cavity may close rapidly by granulation, and in a few days the patient be entirely well; or the abscess may refill. External opening I have never known to take place. It is rare for much burrowing to occur. In young or very delicate infants the constitutional symptoms may be so severe that the child continues to fail even after the evacuation of the abscess, and dies usually from broncho-pneumonia.

Death may occur from asphyxia due to pressure upon the larynx, to œdema of the glottis, or from rupture of the abscess into the air passages, especially if this occurs during sleep. Carmichael, Bokai, and others have reported deaths from ulceration into the carotid artery, or one of its large branches. Carmichael's patient was only five weeks old. The general mortality is from five to ten per cent; many deaths are due to a failure to make the diagnosis. Gautier has collected ninety-five cases, with forty-one deaths. In my experience death has most frequently resulted from late broncho-pneumonia; in one case it was due to a secondary retro-œsophageal abscess.

*Diagnosis.*—Retro-pharyngeal abscess is to be suspected if in an infant there is difficulty in swallowing, noisy dyspnœa, mouth-breathing, and the head drawn backward. A positive diagnosis is possible only by a digital examination of the pharynx. The mistake most often made is, that the physician, called to a young child suffering from great dyspnœa, has jumped at a diagnosis of laryngeal stenosis, and forth-

with performed tracheotomy or intubation, without taking the trouble to get the history or to make a careful examination of the pharynx. Many such cases are reported in which the child has died during the operation or immediately afterward, the autopsy first revealing the nature of the disease. A sudden attack of dyspnœa like that caused by the rupture of an abscess might be produced by the lodgment of a foreign body in the pharynx or larynx. A digital examination would aid in the diagnosis. I once saw in an infant a sarcoma of the pharyngeal lymph glands which gave an external and internal tumour exactly like that of a retro-pharyngeal abscess.

*Treatment.*—Before the abscess has pointed, hot applications may be made to the throat to relieve the symptoms and to hasten the formation of pus, since resolution is not to be expected. Spontaneous opening should never be waited for, on account of the danger of the rapid development of serious symptoms from pressure or œdema, or of suffocation from an opening into the air passages, especially during sleep.

As soon as the diagnosis is made the case should be carefully watched, and as soon as a point of superficial fluctuation is detected, but not before, the pus should be evacuated. External incision has its advocates, but the internal opening is, to my mind, much to be preferred. In opening through the mouth the patient should be seated in an upright position and the head firmly held. The use of a mouth-gag may cause asphyxia. With the finger as a guide, a bistoury, which has been guarded to its point, is introduced and the abscess opened at its thinnest portion, the incision being made toward the median line. The head should then be bent forward, to allow the pus to escape through the mouth. It is well to insert the finger into the cavity to enlarge the opening and break down any septa; for after a simple puncture the abscess may refill. Incision, although usually easy, in some cases may be quite difficult on account of the swelling and the small pharynx of the infant. For the past few years I have adopted the plan of opening these abscesses with the finger nail, sharpened to a point, a procedure simple, efficient, and free from danger. I have seldom seen a case in which this was difficult. The amount of pus evacuated is from one drachm to half an ounce. In the majority of cases no after-treatment is required. The relief of the dyspnœa and dysphagia is immediate, and, except in young infants, recovery usually rapid.

**Retro-pharyngeal Abscess from Pott's Disease.**—This form is rare in comparison with that just described, and under three years of age it is extremely so. These abscesses are usually larger, and the amount of pus contained may be from four to eight ounces. They form very much more slowly, often lasting for months, and as with other secondary abscesses, the constitutional symptoms are seldom severe. The swelling is frequently in the median line, and is not so circumscribed as in the

idiopathic cases. The pus often burrows along the spine for several inches.

The symptoms of Pott's disease of the cervical region are usually present for several months before the appearance of the abscess. Sometimes the abscess precedes the deformity, and it may be the first intimation of the existence of bone disease. The local symptoms resemble those of the idiopathic cases, but they develop more slowly, and sudden attacks of fatal asphyxia are very rare. External swelling is usually seen, and it may be quite large, extending almost from one ear to the other, forming a distinct collar. On digital exploration there may be found an irregularity of the anterior surface of the cervical vertebræ, and occasionally a marked angular prominence.

When left to themselves these abscesses may open externally in front of the sterno-mastoid muscle just below the jaw, sometimes nearly as low as the clavicle; they may rupture internally into the pharynx, the œsophagus, or the air passages; or they may burrow a long distance in front of the spine. Death may result from pressure upon the larynx, or from rupture into the larynx, trachea, or pleura; all these, however, are rare. The abscesses not infrequently refill after they are evacuated, and occasionally a discharging sinus is left for many months.

*Treatment.*—These abscesses should be opened as soon as they are large enough to give rise to local symptoms. The external incision just in front of the sterno-mastoid muscle is generally to be preferred to opening through the mouth, since it gives better drainage, and the after-treatment is more easily carried on; and a sinus opening externally is less objectionable than one opening into the pharynx.

#### ADENOID VEGETATIONS OF THE VAULT OF THE PHARYNX.

This is a very common condition and one much neglected by the general practitioner. It is the source of more discomfort and the origin of more minor ailments than almost any other pathological condition of childhood.

There is a mass of lymphoid tissue situated at the vault of the pharynx which in structure closely resembles the tonsils. It is often spoken of as the "pharyngeal tonsil." Like the faucial tonsils, this may become greatly hypertrophied, so as to form a tumour large enough to fill the rhino-pharynx completely. These tumours have a broad attachment which is sometimes more to the roof, and sometimes more to the posterior wall of the pharynx. The term *adenoid vegetations* was given to them by Meyer, who first described them in 1868. In infancy these growths are soft, vascular, and spongy; in older children they become firm, dense, and more fibrous. Their appearance is well shown in Fig. 47. Adenoid vegetations are associated with hypertrophy of the faucial

tonsils in about one-third of the cases. Growths large enough to cause decided nasal obstruction may in time produce changes in the facial bones amounting to positive deformity. The bony palate may be dome-shaped or even acutely arched; the dental arch of the upper jaw be-

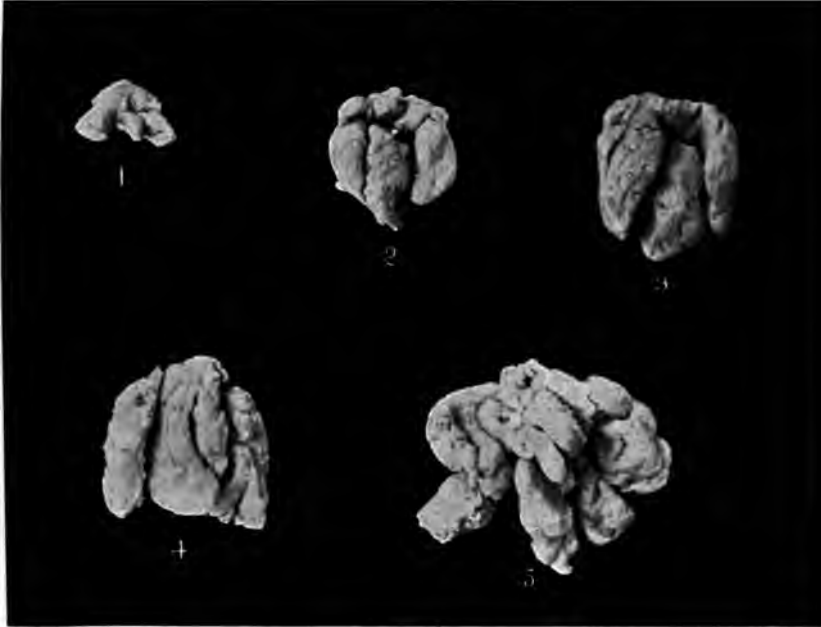


FIG. 47.—ADENOID VEGETATIONS, NATURAL SIZE. (1) From child eight months old; (2) from child twenty-two months old; (3) from child two and one-half years old; (4) from child two and one-half years old; (5) from child three years old. With the exception of (5) all were removed with a single sweep of the curette. Although the growths represented are somewhat larger than the average for the ages mentioned, just such ones are constantly met with in practice.

comes almost V-shaped. Deformities of the thorax also occur, which will be described with the symptoms.

**Etiology.**—Hereditary influences certainly play some part in the production of this condition. I have frequently known every one of a large family of children to be affected, and often the parents have suffered from the same condition. In many cases adenoid growths are congenital. Rachitic children are somewhat oftener affected than others, but no connection with syphilis has been traced. Much interest has lately been awakened regarding the relation of adenoid growths to tuberculosis. Of 945 cases collected by Lewin in which specimens of adenoids were examined, tuberculosis was present in five per cent. Though this proportion is no doubt much higher than will be found in private practice, the fact is an important one; for it is highly probable that this is the channel of infection in not a few cases of tuberculosis. Adenoids

are most common in damp, changeable climates. Their first symptoms often follow an attack of measles, scarlet fever, or diphtheria. The repeated head colds are more often a result than a cause of the condition.

**Symptoms.**—The symptoms of adenoid growths are usually first noticed when children are from eighteen months to three years old; but they may be present almost from birth. I have in several instances seen them to a marked degree in infants only a few months old. The symptoms generally increase in severity as age advances, being always better in summer and worse in winter, until the age of six or seven is reached. The chief symptoms are those which relate to (1) chronic rhino-pharyngeal catarrh, (2) mechanical obstruction, (3) otitis and other aural conditions, (4) general malnutrition and anæmia, (5) reflex nervous phenomena.

The rhino-pharyngeal catarrh shows itself by a persistent nasal discharge, frequently recurring acute attacks, or head colds, during the entire winter season. In susceptible children these attacks are often the beginning of a bronchitis, which may keep a young child indoors almost the entire winter.

The obstructive symptoms are inability to blow the nose, mouth-breathing constantly or only during sleep, and a nasal voice. The difficulty in breathing is increased when the child lies upon the back. In consequence of this, children sleep in all sorts of positions—lying upon the face, sometimes upon the hands and knees, and often toss restlessly about the crib in the vain endeavour to find some position in which respiration is easy. The attacks of dyspnoea at night may amount almost to asphyxia, and are the explanation of many of the so-called night-terrors from which children suffer. When the obstruction has existed from infancy there are often deformities of the chest: these are most marked in rachitic subjects. The most frequent one consists in deep lateral depressions of the lower part of the chest, with a prominence of the sternum. The deformity is due to interference with pulmonary expansion.

There is often seen a flattening at the root of the nose, and sometimes a prominence of the transverse vein in this region.

Some impairment of hearing exists in a large proportion of the cases. Blake (Boston) found this to be true in 39 out of 47 cases examined; in 35 of these marked improvement in the hearing followed removal of the adenoid growths. Deafness may be due to tubal catarrh or to otitis. Often a history is given of several attacks of suppurative otitis.

The reflex symptoms associated with adenoid growths are many. One of the most important is catarrhal spasm of the larynx, or the familiar spasmodic croup. In my experience the majority of young children who are subject to such attacks have adenoids, the removal of which is frequently followed by their complete cessation. Other respiratory

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symptoms associated with adenoids are intractable coughs, frequently of a spasmodic character, without bronchial symptoms or signs, and persistent hoarseness, lasting for months or even years, and recurring every cold season. Both these conditions are often cured by the removal of the adenoids after all other treatment has been without effect. To these growths bronchial asthma also is very frequently due. Their relation to incontinence of urine is often an intimate one; the two coexist in a large number of patients, and in a certain number removal of the adenoids cures the incontinence. Headaches are very common; stammering may be present; chorea and even epileptiform seizures have been attributed to adenoids, although I have never seen either.

The general health of patients suffering from adenoids may be impaired from lack of oxygen due to obstructed respiration, from loss of sleep, and from confinement to the house, necessitated by attacks of bronchitis or head colds. Marked anæmia is often present. In old and neglected cases of a severe character, children may be stunted in growth, and their facial expression dull and stupid. They are languid, listless, often depressed, and this with their deafness frequently causes them to be regarded in school as children who are somewhat deficient mentally.

These patients are always better in summer and worse in winter. The natural course of the growths if left to themselves is to increase up to a certain point, and then to remain stationary until puberty, when they usually undergo atrophy. This, with the marked increase in the capacity of the rhino-pharynx which occurs at this time, results in a disappearance of the most aggravated symptoms. The removal of the patient to an elevated region with a dry atmosphere will often result in a relief from all the symptoms, and a diminution in the size of the growth, but unless such a change in residence is permanent the symptoms are liable to return. Under ordinary circumstances there is little or no tendency to spontaneous recovery. In children with adenoid growths attacks of diphtheria, scarlet fever, measles, and whooping-cough are all likely to be more severe.

**Diagnosis.**—In a well-marked case the condition is usually evident from the history, and can scarcely be overlooked. The intractable nasal catarrh, upon which no treatment, local or general, has more than a temporary influence, the mouth-breathing, the disturbed sleep, and the slight deafness—all are characteristic. In some even of the marked cases, attention may be drawn to the larynx, bronchi, or ears as the seat of disease. At other times the patients come for treatment on account of the general symptoms—the nervous depression, the headaches, or the anæmia. In rare cases the leading symptom may be epistaxis. The symptoms do not always depend upon the size of the growth, for in a small throat quite a small growth may cause very marked symptoms.

Although the history is in most cases clear, only an examination can



make us certain that an adenoid growth exists. The best method of examination consists in a digital exploration of the pharynx; but this requires a little practice before it is very satisfactory. The head is steadied by one hand, and the forefinger of the other is passed up behind the soft palate. The growth is ordinarily felt as an irregular, granular, soft, velvety mass, or sometimes as a firm tumour completely blocking the passage; and the finger, when withdrawn, is almost invariably covered with blood. By posterior rhinoscopy, the growth in older children can often be seen.

**Treatment.**—The disappearance of adenoid growths is possible only when they are small. This is aided by removal to a warm, dry climate for the winter season. All possible means should be employed to prevent these patients from taking cold, such as proper clothing, cold sponging, cod-liver oil, etc. With the larger growths these methods may improve the catarrhal symptoms, but can hardly affect the obstructive ones. The reduction of tumours of any considerable size by local applications is, I think, a delusion; every marked case that has come to my notice has been relieved only by operation.

Removal of adenoid growths is indicated: (1) When the obstructive symptoms—habitual mouth-breathing, disturbed sleep, nasal voice, chest deformities, etc.—are marked; (2) for a chronic nasal discharge, constantly recurring head colds, particularly when these tend to attacks of bronchitis or laryngitis; (3) where there is asthma or repeated attacks of catarrhal spasm of the larynx; (4) with deafness, chronic otitis, or repeated attacks of acute otitis; (5) for certain nervous symptoms—enuresis, stammering, chorea, headaches, night-terrors, etc. Although striking improvement is not infrequent, one should be cautious about promising too much from operation, especially as regards the nervous conditions; also in older children when there is deafness or asthma.

The preferable time for operation is the late spring or early summer, in order that during the warm months the mucous membranes may have an opportunity to regain their normal condition; however, operation may be done at any time except during attacks of acute catarrh. Unless the symptoms are very marked, I prefer to defer operation until a child is at least two years old.

Removal of adenoids by scraping with the finger nail is at best a very uncertain method, and is not to be advised, except in the case of children under two or two and a half years old, where the growths are generally small and the patients easily handled. The operation is preferably done with general anaesthesia: first, for the sake of thoroughness; secondly, to avoid the fright and pain which so bloody an operation is apt to cause in those who are older, and especially in very nervous children. So many deaths from operations for adenoids or tonsils under chloroform have now been reported, and so many narrow escapes have occurred that

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have not been reported, that chloroform anæsthesia should, I think, be given up altogether. My preference is for ether; in older children it may with advantage be preceded by nitrous oxide, and sometimes with such patients the nitrous oxide alone may be used; but this is not to be advised with very young children. Deep anæsthesia is not usually necessary, and if the semi-erect position is assumed it increases the danger of the entrance of blood or portions of the growth into the larynx, which might cause asphyxia.

The only instruments required are a mouth-gag, like that used for intubation, and modified Gottstein's curettes, which should be sharp. The physician should have several sizes with different curves to suit the size and attachment of the growth and the capacity of the throat. Many of the instruments used for young children are too large, the smaller ones being more easily manipulated and less liable to do harm.

During operation it is an advantage to have the patient raised to a little more than a half-reclining posture and the head firmly steadied. Many of the growths encountered in ordinary practice, such as Nos. 1, 2, and 3 in the illustration, can be removed with one sweep of the curette, the mass usually coming away in a single piece. Others may require the instrument to be used two or three times. The forceps (Lowenberg's and various modifications) in unskilled hands are capable of doing much harm. One unfamiliar with their use may easily tear away pieces of the uvula, soft palate, pharyngeal wall, and even portions of the Eustachian tubes.

Hæmorrhage is always abundant, and seems alarming to one who sees it for the first time. In an average case it amounts to one or two ounces, but generally ceases in a few minutes. A child should not pass from the physician's observation until all bleeding has stopped. He should be kept quiet, preferably in bed, for twenty-four hours; and in the house for five or six days, unless the weather is warm. No after-treatment is necessary. Recurrences are occasionally seen even after a thorough operation by an experienced person. But many of them are due to the fact that the primary operation was incomplete. The improvement generally begins in a few days, sometimes at once, though the full benefit may not be seen for a month. The breathing becomes freer, the sleep more quiet; the mouth may soon be habitually closed; voice and hearing improve, and the benefit to the general health is soon apparent. The pallor, listlessness, and inattention disappear, and a rapid increase in weight often follows. The entire appearance of the child may in a few months be transformed.

**Dangers and Accidents from Operation.**—While it is rare that any accidents of a serious nature are met with, it should not be forgotten that they may occur. Undue laceration of the parts may result from a bungling operation, particularly with too large curettes or with the for-

ceps. Hæmorrhage may be excessive or even fatal. I have seen but one case of fatal hæmorrhage, this in a bleeder, and but one other instance of serious hæmorrhage. A fatal result is exceedingly rare. Hæmorrhage may be continuous after operation, or secondary, in which case it almost invariably occurs within twenty-four hours. It is important, therefore, that the patient be kept under observation for that time. Bleeding is best controlled by injecting into the rhino-pharynx through the nostrils one or two drachms of hydrogen peroxide, full strength, or, this failing, a solution of adrenalin (1-1000) may be used in the same manner. As a last resource plugging of the rhino-pharynx and posterior nares may be resorted to. In all cases the patient should be kept absolutely quiet.

Occasionally an acute attack of bronchitis or otitis occurs after operation; and in a few recorded instances acute meningitis has followed. The danger of asphyxia from the entrance of blood or the tumour into the larynx has already been mentioned.

The danger from chloroform anæsthesia is due not so much to the nature of the operation as to the condition of the patient. It is now well established that all children in whom the condition known as status lymphaticus is present, bear chloroform very badly.

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### CHAPTER III.

#### *DISEASES OF THE TONSILS.*

THE tonsils are lymphoid structures closely resembling Peyer's patches, but, instead of having a flattened surface, the lymphoid tissue in the tonsil is folded upon itself, forming quite deep depressions—the tonsillar crypts. These crypts, like the surface of the tonsils, are lined by epithelial cells. They contain lymphoid cells, desquamated epithelium, particles of food, and bacteria. Under normal conditions the tonsils take no part in absorption from the mouth. When, however, their epithelium is diseased or removed, the tonsils absorb with very great facility every sort of poison which the mouth may contain. Such poisons are taken up by the lymphatics, and through them reach the general circulation.

Acute inflammation of the tonsils, like that of the pharynx, occurs regularly in diphtheria, scarlet fever, and measles, less frequently in the other infectious diseases. The secondary forms will be considered with the diseases with which they are associated.

Acute catarrhal tonsillitis, or inflammation of the mucous membrane covering the tonsils, occurs as part of the lesion in acute pharyngitis, but very rarely is seen alone.

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## MEMBRANOUS TONSILLITIS.

(*Pseudo-diphtheria; Streptococcus Angina; Croupous Tonsillitis.*)

This occurs both as a primary inflammation and secondary to the acute infectious diseases, especially scarlet fever and measles. The angina of scarlet fever is essentially a part of that disease and is more fully considered in connection with it.

**Etiology.**—As was first shown by Prudden in 1888, and abundantly confirmed by others since that time, this inflammation is usually due to the streptococcus; it may be found alone, or associated with the staphylococcus aureus, and occasionally the staphylococcus may be found alone.

The streptococcus is very frequently found in the throats of healthy children, particularly in winter and in cities, and more often in those who live in tenements or who are inmates of hospitals or other institutions. The local conditions in the mucous membranes during an attack of measles, scarlet fever, and other infectious diseases, are especially favourable for the development of these germs, which at such times are very often present in great numbers even when no membrane is seen.

**Lesions.**—In the primary cases the membrane is generally confined to the tonsils or is chiefly there, only small deposits appearing elsewhere. In the secondary cases, the entire pharynx may be covered and the disease may extend to the nose, the mouth, the middle ear, and rarely to the larynx, trachea, and bronchi.

The structure of the membrane resembles that of true diphtheria, and it may be impossible by a microscopical examination to separate the two diseases.

In the mild cases the inflammation of the mucous membrane is a superficial one and the pseudo-membrane is not very adherent. In the severe cases, chiefly the secondary ones, the process extends much deeper. Besides the pseudo-membrane upon the surface, there is intense congestion, œdema, and cell-infiltration of all the lymphoid and cellular tissue of the pharynx. It may involve the tonsils, soft palate, uvula, epiglottis, adenoid tissue of the vault and the entire pharyngeal ring, and also extend to the external lymph nodes and surrounding cellular tissue. The process both in the throat and externally in the neck may terminate in resolution, suppuration, or in necrosis.

The streptococci are found in the false membrane, in the underlying mucous membrane, in the lymph spaces and in the lymph nodes. In the most severe cases there are present the lesions of a general streptococcus infection.

**Symptoms.**—1. *The Primary Cases.*—The onset is usually abrupt, with well-marked symptoms: there are frequently chilly sensations, headache, vomiting, general pains, and in most cases the child complains of soreness of the throat and pain on swallowing. There are first seen a

general redness and swelling of the tonsils, sometimes of the entire pharynx; shortly afterward membranous patches appear upon the tonsils. These vary greatly in appearance. In colour they are yellow or gray, often changing later to a dirty olive tint. (Plate XVIII, c.) The membrane seems loosely attached and can frequently be wiped off with a swab. It is often irregular in its outline, which is not sharply defined. The membrane usually remains but three or four days and disappears rapidly. As a rule, it is limited to the tonsils, and does not spread after it first forms. Occasionally, however, small patches are also seen upon the fauces or the pharynx. The constitutional symptoms are generally severe during the first two days, and the temperature may be 103° or 104° F., but by the third day it falls, and most of the symptoms subside. It is rare for the disease to extend either to the nose or the larynx. Generally there are no complications and no sequelæ.

2. *The Secondary Cases.*—Some of these are mild, and do not differ from those just described, but most of the severe cases are included in this group. The clinical picture of the latter is that of *scarlatina anginosa*, as given by the older writers.

In measles the throat symptoms are somewhat later than in scarlet fever; they may begin at the height of the primary fever, and increase while the eruption fades. The process is almost invariably complicated by broncho-pneumonia.

Secondary cases as a class are characterised by high temperature (Fig. 48), rapid, feeble pulse, great prostration, delirium, apathy or stupor, and often albuminuria. In fatal cases death usually occurs at the height of the disease, from asthenia, broncho-pneumonia, or nephritis. If none of these complications develop, patients may withstand the toxic symptoms even when they are very severe.

There may be in connection with the local process in the throat, deep sloughing of the tonsils or adjacent structures, suppuration of the lymphatic glands or in the cellular tissue of the neck, occasionally followed by serious hæmorrhage. However, these complications are rare, and if the patient survives the danger of the acute stage of the disease, he usually recovers.

**Diagnosis.**—The clinical features which distinguish membranous tonsillitis from diphtheria are considered under the latter disease. It is impossible in any case to be certain of the diagnosis except by cultures; for, although by clinical symptoms alone one may in the great majority of cases be certain that a given case is one of true diphtheria, to say that any membranous inflammation of the throat is not diphtheria is impossible. The bacteriologists have taught us to be cautious in pronouncing too positively even upon mild cases, as it has been shown that some of them may be caused by most virulent diphtheria bacilli.

A membrane which appears in the throat early in the course of

measles or scarlet fever, or at the height of the primary disease, is usually due to the streptococcus; while one which develops late or after the primary fever has subsided, is frequently due to the diphtheria bacillus.

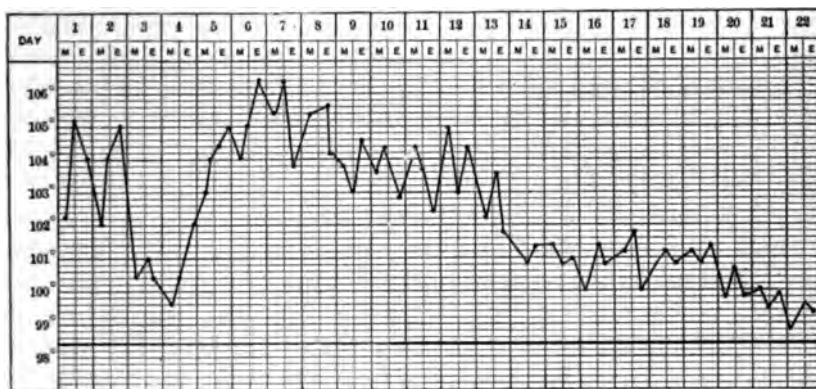


FIG. 48.—STREPTOCOCCUS ANGINA, FOLLOWING MEASLES. The chart begins at the time of the full eruption in a severe case of measles. On the third day the temperature fell, with fading eruption, and child seemed convalescent. With secondary rise in temperature, the tonsils, which before had been only red, showed membranous patches, the exudation rapidly spreading until the entire pharynx was covered; throat symptoms very severe, with great swelling of cervical glands, but the membrane did not extend beyond the pharynx. From sixth to twelfth day a most profound septicæmia, so that life was despaired of. The patient was a vigorous child, and, escaping both nephritis and pneumonia, made a good recovery. Convalescence quite rapid; no sequelæ. Repeated cultures were made from the throat, but all showed only streptococci. Patient a girl four years old. Case observed in private practice.

**Prognosis.**—In a child previously healthy, primary membranous tonsillitis is not a serious disease. In the secondary cases, we find very different conditions. From the best available statistics it would appear that the usual mortality, when it is secondary to scarlet fever and measles, is from fifteen to twenty per cent. However, when these diseases prevail epidemically in institutions, the mortality is often higher than this.

**Treatment.**—Every child with a membranous patch on the tonsils requires close watching; strict quarantine should be enforced until the diagnosis is positively settled. If under three years old, unless the case can be seen frequently, diphtheria antitoxin should be administered, pending the result of a bacteriological examination. The primary cases require only the treatment of an attack of tonsillitis.

In the severe secondary cases the nose and pharynx should be syringed with a warm saline solution every two hours by day and every four hours by night. Where the swelling and œdema are great, benefit may result from frequent spraying with solutions containing adrenalin, also from inhaling hot vapour impregnated with eucalyptol, benzoin, etc. As an

- external application, whenever there is great adenitis and cellulitis, nothing is so beneficial as the ice-bag.

The general management of these cases as to feeding, stimulants, etc., is the same as in diphtheria. Aside from stimulants no internal medication should be attempted with young children. Those who are older may take with advantage tr. ferri chlor., gtt. v to x, with glycerin, every three or four hours.

### ULCERO-MEMBRANOUS TONSILLITIS.

(*Vincent's Angina.*)

This is an inflammation somewhat resembling croupous tonsillitis, but it is often unilateral and associated with superficial ulceration. The tonsil is covered with a dirty yellowish exudation, which may be mistaken for diphtheria. There is superficial necrosis, and when this tissue is wiped away with a swab, bleeding occurs. The disease is further distinguished by the swollen lymph nodes at the angle of the jaw, and by the fact that the constitutional symptoms which accompany other forms of tonsillitis are either very slight or absent altogether. The etiology is similar to, if not identical with, ulcerative stomatitis, with which it is sometimes associated. At such times the breath is foul and there is often profuse salivation.

Ultero-membranous tonsillitis was first described by Vincent, and by him attributed to a fusiform bacillus which he described, although a spirillum was found associated with it. Vincent's observations have been confirmed, and it has been shown that the spirillum is a degenerative form of the bacillus.<sup>1</sup>

The chief interest in ultero-membranous tonsillitis lies in the diagnosis, although it is not an infrequent disease. It is to be treated, like ulcerative stomatitis, by the internal administration of chlorate of potash, combined with the local application of some antiseptic, such as peroxide of hydrogen or a ten-per-cent solution of nitrate of silver.

### FOLLICULAR TONSILLITIS.

This is the most frequent and most characteristic form of inflammation of the tonsil. It is essentially an inflammation of the tonsillar crypts, and secondarily of the whole glandular structure.

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<sup>1</sup> Vincent's bacillus is about twice as long as the Klebs-Loeffler bacillus. It is thin, with pointed ends, and sometimes bent; it is negative to Gram's stain. The fusiform bacillus is occasionally found alone; the spirillum, never alone. The bacillus is found in smears from the affected tonsil, in making which it is recommended to go deeply into the necrotic tissue, since the superficial parts are crowded with other bacteria. It is grown with difficulty and only upon special culture media.

**Etiology.**—There is seen in certain children a predisposition to attacks of tonsillitis, so that from very slight exciting causes these occur—sometimes from exposure, sometimes possibly from derangement of the stomach, and sometimes without any evident reason. Children with a rheumatic inheritance appear to be more susceptible than others. One attack predisposes to a second. Patients suffering from chronic hypertrophy of the tonsils are exceedingly prone to acute tonsillitis. It is not very common in infancy, but after this period it is very frequent throughout childhood. The disease, in all probability, begins as an infectious inflammation at the bottom of the crypts, due to the presence of streptococci or staphylococci, which readily enter from the mouth, and excite an attack whenever favourable conditions are present.

**Lesions.**—As a result of the inflammation, the tonsillar crypts are filled with epithelial cells, pus cells, mucus, and bacteria. These form masses which appear at the mouth of the crypts as small yellow dots, often mis-called ulcers. Sometimes, in addition, fibrin is poured out, and forms, with the other inflammatory products, little plugs which project somewhat from the surface of the mucous membrane, and which can easily be pressed out. Accompanying the changes in the mucous membrane above mentioned, there are acute congestion and swelling of the whole tonsil, with more or less proliferation of the lymphoid tissue. Follicular tonsillitis is almost always bilateral. Although the pathological process is generally limited to the tonsils, there may be more or less pharyngitis associated.

**Symptoms.**—The general symptoms usually appear before the local ones, and are often quite severe. The onset is abrupt, with chilly sensations, occasionally a distinct rigor. In infants there is often vomiting, and sometimes diarrhoea. There is pain in the back, in the muscles of the extremities, and in the head. Sometimes there is pain in the lateral cervical muscles. The temperature rises rapidly to 102° or 103° F.; often it touches 104° or 105° F.

The first local symptoms are some swelling of the tonsils and the appearance upon them of isolated yellow spots a little larger than a pin's head. Often these can be wiped off with a swab, or the little plugs can be squeezed out, leaving slight depressions. Later there is acute congestion of the tonsil, with more swelling. Even when the disease is at its height the local pain and discomfort may be only moderate, and in many cases scarcely noticeable. The swelling and tenderness of the lymph glands behind the angle of the jaw are not great, and may be absent.

The constitutional symptoms, as a rule, last three days, and are most severe upon the first day. The local symptoms last somewhat longer, but usually by the end of the fourth day the exudate has disappeared, although enlargement of the tonsil may persist for a week or even longer. On account of the connection of tonsillitis with rheumatism, the heart



should be watched during attacks, especially in those who are subject to them.

**Diagnosis.**—Tonsillitis may be confounded at its onset with scarlet fever. The great frequency of tonsillitis makes inspection of the throat imperative in every case of acute illness in children. The diagnosis from diphtheria is considered in connection with that disease.

**Treatment.**—Follicular tonsillitis is a mild disease without danger to life, and one which runs a short, self-limited course. The indications are, therefore, to make the patient as comfortable as possible by the relief of individual symptoms. Older children, particularly those who are rheumatic, should be treated with sodium salicylate, or aspirin, four or five grains every three hours being given for the first twenty-four hours, and later less frequently. To infants these drugs must be given in smaller doses and with care, lest they upset the stomach. The general muscular pains of the first day are best relieved by phenacetine, two grains every four hours to a child three years old. Later it may be used in smaller doses, but enough should be given to make the patient comfortable.

Local treatment is better omitted with infants. Older children may gargle with a solution of boric acid or may use a spray of Dobell's solution. Benefit often follows painting the tonsils with tincture of iodine or a ten-per-cent solution of silver nitrate. In all doubtful cases the patient should be isolated and the same general treatment adopted as in diphtheria.

#### PHLEGMONOUS TONSILLITIS—PERITONSILLAR ABSCESS—QUINCY.

This is an inflammation of the cellular tissue surrounding the tonsil, sometimes invading the tonsil itself. It may terminate in resolution, but usually goes on to the formation of an abscess. Phlegmonous tonsillitis is much less common in children than in adults, and, compared with the other forms, it is a rare disease in early life. It is the only variety which is regularly unilateral. In most cases the inflammatory process is circumscribed, but in rare instances there is seen a diffuse phlegmonous inflammation of the pharynx.

In certain patients there exists a constitutional predisposition to the disease, which may be associated with rheumatism. The exciting cause may be exposure, or anything which may reduce the patient's general health, to which there is added local infection. Catarrhal pharyngitis predisposes to this disease.

**Symptoms.** The onset resembles that of follicular tonsillitis, the temperature is often high, and the muscular pains and prostration severe. There is very severe pain in the throat, which is increased by deglutition, and finally may be so great that swallowing is almost impossible. It is difficult to open the mouth. There is pain in the neck, and swelling of the

neck, and often tenderness. In the beginning but little can be seen on inspection, even though the patient complains of a very sore throat. This is always a suspicious circumstance, and should lead one to look out for quinsy. It is due to the fact that the inflammation begins in the deeper tissues, and that the mucous membrane is affected later. After twenty-four or forty-eight hours there is usually quite marked swelling, which is rather more behind the tonsil than elsewhere, pushing it upward and forward; sometimes it is more in front of the tonsil. A little later there is intense inflammation of the mucous membrane covering the tonsil, fauces, and uvula, and not infrequently a fibrinous exudate; the uvula may be pushed to one side, and the isthmus of the fauces diminished to less than one-half its natural size. In one of my own cases marked torticollis was present, and existed for two or three days before the diagnosis of quinsy could be made by the other symptoms.

In most cases the recognition of quinsy is quite easy by attention to the symptoms above mentioned. By inspection of the throat, less information is sometimes obtained than by palpation; by this means a fulness, and later a point of fluctuation, can readily be made out. Acute phlegmonous tonsillitis generally involves no danger to life. In very young infants serious results may follow spontaneous rupture during sleep; and in older children occasionally there may be œdema of the glottis. If not treated, abscess usually forms in from five to seven days, and opens spontaneously.

**Treatment.**—If an early diagnosis is made an attack of quinsy may possibly be aborted. For this many drugs have been advocated, but to my mind the best is salol, which should be given in doses of two grains every two hours to a child of five years. In some patients larger doses may be used. This may be combined with small doses (gr.  $\frac{1}{4}$ ) of Dover's powder. Relief may be afforded by very hot or cold applications, according to the sensations of the patient. The holding of ice in the mouth and the application of an ice-bag externally, often give great comfort. In other cases, gargling with very hot water and the application of hot flaxseed poultices externally, will be preferred. As soon as fluctuation is detected an incision should be made with a guarded bistoury. If made too early, only a small amount of pus is evacuated and the abscess may refill. After spontaneous rupture the relief to symptoms is usually immediate.

#### CHRONIC HYPERTROPHY OF THE TONSILS.—CHRONIC TONSILLITIS.

The condition known as chronic hypertrophy is a permanent enlargement due to a proliferation of the lymphoid tissue of the tonsils, and an increase in the connective-tissue stroma. If the increase in the connective tissue is slight, the tonsil is soft; if it is great, the tonsil is firm

and hard, almost like a fibrous tumour. All degrees are found. Associated with hypertrophy of the tonsils there are usually found adenoid growths of the pharynx, both of these depending upon similar local and constitutional conditions. There is in nearly all marked cases a chronic pharyngeal catarrh which may involve the Eustachian tubes.

**Etiology.**—Hypertrophy of the tonsils is an exceedingly common condition in the cities of the seacoast and lake districts of the temperate zone. In a routine examination of 2,000 New York school children, Chappell found enlargement of the tonsils sufficiently marked in 270 cases to be considered pathological. The causes are constitutional and local. The condition frequently exists in certain families for several generations. It is not connected with tuberculosis. It occurs in children who are in other respects healthy. Hypertrophy of the tonsils is often a congenital condition, increasing slowly during infancy, so as to produce marked symptoms by the time the child is two years old. The most important of the local causes are attacks of acute or subacute pharyngitis. While it is true that attacks of acute inflammation are often the cause of hypertrophy, it is also true that hypertrophy is one of the most frequent predisposing causes of acute attacks, and that it may be seen in children who have never had tonsillitis.

**Symptoms.**—Hypertrophy of the tonsils is rarely marked enough to cause any decided symptoms before the end of the second year, although I once saw in a younger child enlargement sufficient to bring the two tonsils into contact. The most important local symptoms, formerly ascribed to hypertrophied tonsils, are now known to depend upon adenoid growths of the pharynx. As these conditions are so frequently associated, it is somewhat difficult to determine which symptoms are due to the tonsils alone. In a marked case, the most prominent symptoms are mouth-breathing, disturbed sleep accompanied by snoring, and nasal voice; the patient in some cases talking as though he had food in his mouth. There may be some difficulty in swallowing solid food. Enlarged tonsils may often be felt externally. As a consequence of the obstruction of the Eustachian tubes there may be deafness. Deformities of the chest, such as pigeon-breast, are occasionally seen, but probably depend more upon obstructed respiration by adenoids than by the tonsils.

The soft tonsils may diminish somewhat in size spontaneously. They sometimes shrink very decidedly after an attack of acute tonsillitis, scarlatina, or diphtheria. As a rule, the tonsils become firmer and harder as time passes. They usually increase in size up to a certain point, and then remain stationary, or diminish slightly in size, when they are removed surgically. During the course of cases of inflammation of the tonsils, the tonsils may become so large as to perpetuate a general catarrh of the pharynx, and in such cases the spontaneous improvement is to be looked for, being aided.

**Treatment.**—Very large tonsils are a source of continued danger to the patient, and in every case of marked hypertrophy treatment should be advised. The danger may be from Eustachian catarrh and deafness, or from repeated attacks of acute tonsillitis. But quite as important as these is the fact that they increase the liability to contract diphtheria, and add to the dangers both from diphtheria and scarlet fever. If the patient is removed from the locality in which acute tonsillitis is liable to occur, to a dry climate, considerable improvement is likely to result in a young child in whom the tonsils are soft, but not much is to be expected in older children with hard, fibrous tonsils, except, perhaps, a cure of the accompanying pharyngeal catarrh.

No internal remedy offers much chance of benefit. Astringent applications may accomplish something in recent, but practically nothing in old cases. In every marked case, operation is the only thing which can be relied upon to effect a cure. For convenience of consideration, the cases may be divided into three groups: (1) Those in which the tonsils are nearly or quite in contact; (2) those in which they project only slightly beyond the faucial pillars; (3) those in which the tonsils, although large, are deeply imbedded. All of the first group should unquestionably be operated upon, unless the patient's general condition is such as to forbid operation of any kind. In the second group operation is not indicated unless there are repeated acute attacks, or the tonsils are the seat of chronic infection. Whether an operation is done in the third group will depend upon the individual case. If there are frequent attacks of acute tonsillitis or evidence of involvement of the ears operation should be performed.

Various operations are in use for the removal of hypertrophied tonsils: the wire snare, amputation with the guillotine, and enucleation. Each has its advocates and each its advantages. The use of the snare is accompanied with little risk of hæmorrhage. It is a painful operation, some preliminary dissection is usually required, and hence general anæsthesia is necessary. Amputation by the guillotine is simpler and for well-projecting tonsils quite sufficient. The risk of hæmorrhage in children is slight. An anæsthetic is unnecessary if only the tonsils are to be removed. The amount of shrinkage from cicatrization after operation has been, in my experience, generally less than was expected. Enlargement of the tonsil subsequent to amputation is sometimes seen, oftener if the patient operated on is under two years old. I am not yet convinced of the advantages of complete enucleation, now much in vogue, as a routine operation for hypertrophied tonsils, but in certain cases nothing else is adequate. Such are the broad, deeply imbedded, adherent tonsils. Excessive hæmorrhage after any form of operation may be controlled by digital pressure, or by the application of styptic cotton upon a swab; in extreme cases, by transfixing the tonsil stump

with a harelip pin and the application of a ligature. I have more than once seen physicians greatly alarmed at the gray wound on the day following tonsillotomy, the appearance being such as to lead in several cases to the diagnosis of diphtheria. It is seldom that any but good results follow the operation of tonsillotomy if properly performed. When adenoids of the pharynx are also present, the symptoms may depend more upon them than upon the enlarged tonsils, and little benefit is seen unless the adenoids also are removed.

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## CHAPTER IV.

### *DISEASES OF THE ŒSOPHAGUS.*

#### MALFORMATIONS.

CONGENITAL anomalies of the œsophagus are often associated with those of the lower part of the respiratory tract.

There may be, (1) Congenital fistula of the neck, due to a want of closure between the second and third branchial arches. This gives an external opening just above and to the outside of the sterno-clavicular articulation, which communicates with the upper part of the œsophagus or the lower part of the pharynx. (2) The œsophagus may be absent, the pharynx ending in a blind pouch. (3) The œsophagus may be obliterated in certain portions, being represented only by a fibrous cord. (4) There may be stenosis and dilatation or diverticula. (5) There may be fistulous communication with the trachea, existing either alone or associated with some of the other deformities mentioned.

Congenital narrowing of the œsophagus and fistula of the neck are amenable to surgical treatment. The cases of complete obstruction in the œsophagus are almost of necessity fatal, the patients dying from inanition two or three days after birth.

The symptoms of œsophageal obstruction are regurgitation on attempts at swallowing and the impossibility of passing the stomach tube. An X-ray picture after the administration of bismuth often gives valuable information.

#### ACUTE ŒSOPHAGITIS.

It is quite remarkable, considering the frequency of pathological processes in the pharynx, that these so rarely extend to the œsophagus. Thrush, when very extensive in the pharynx, may involve the upper part of the œsophagus; but there it gives rise to no new symptoms. Diphtheria of the pharynx may invade the œsophagus, but this is seen only in rare instances. Diphtheria of the œsophagus produces no symptoms by which it can be diagnosticated during life.

**Catarrhal Œsophagitis.**—Catarrhal œsophagitis is very rarely met with. It may be caused by lacerations due to swallowing a foreign body, which may excite a simple catarrhal inflammation, or, if the foreign body is sharp and angular, lacerations may be produced which result in ulcerations of variable depth. The chief symptoms of catarrhal œsophagitis are soreness and pain on swallowing. These lacerations, when slight, are healed in a few days, and are rarely followed by any after-effects.

**Corrosive Œsophagitis.**—This is altogether the most frequent form, and the only one which is of clinical importance. The usual causes are the same as of corrosive gastritis, viz., the swallowing of caustic alkalis or strong acids. It is often in the œsophagus that the most extensive injury is done. The effects are superficial or deep, according to the amount of the irritant swallowed and its degree of concentration. There may be simply a destruction of the epithelial layer, which is followed by no serious consequences, or the mucous membrane may be destroyed and the submucous coat invaded; rarely, however, does the injury extend to the muscular layer. If the patient survives the dangers incident to the irritant poisoning and the acute inflammation which follows, healing by granulation and cicatrization takes place, the contraction of the cicatrix gradually narrowing the lumen of the œsophagus until stricture is produced.

The early symptoms of corrosive œsophagitis are mingled with those of inflammation of the mouth, pharynx, and stomach. There is a burning pain in the parts, great thirst, and spasm of the œsophagus on attempts at swallowing. There follows a period of acute inflammation of several days' duration, with great dysphagia and pain, in which the principal danger is œdema of the glottis. After this the patient may be comparatively well until the symptoms of stricture begin, usually in from three to six months after the injury.

The indications for treatment in the early stages are, to neutralise the caustic in order to prevent if possible its deep action, to give oils, demulcent drinks and ice for the local effect, and morphine for the pain.

The treatment of œsophageal stricture is purely surgical.

#### RETRO-ŒSOPHAGEAL ABSCESS.

Acute retro-œsophageal abscess occurs in infancy, though very rarely, the pathology being the same as in acute retro-pharyngeal abscess, the difference being merely one of location. A striking case of this kind occurred in the New York Foundling Hospital. An infant six months old was admitted with high fever (104° F.), severe dyspnoea, but with no loss of voice, which were the prominent symptoms until death occurred four days later. There was a leucocytosis of 100,000. At autopsy an

abscess was found containing about three ounces of pus between the œsophagus and the spine, extending from the larynx to below the bifurcation of the trachea. Shortly afterward I saw a very similar case at the Babies' Hospital, following a retro-pharyngeal abscess which had been opened two weeks before. Similar abscesses have also been observed after acute pharyngitis with the acute infectious diseases.

Retro-œsophageal adenitis, or enlargement of the lymph nodes in this situation without suppuration, is also rare. I once met with a case of this sort in which the gland formed a tumour nearly an inch in diameter at the upper part of the œsophagus, causing pressure symptoms necessitating tracheotomy. The growth was at first thought to be malignant, but completely disappeared after a summer in the country.

Retro-œsophageal abscess may result from the breaking down of tuberculous lymph nodes in the posterior mediastinum, and may give rise to symptoms like those which result from an abscess due to Pott's disease.

Perforation of the œsophagus and a food-fistula connecting the œsophagus and the trachea may result from ulceration caused by a tracheal canula or by a foreign body. This may be accompanied by abscess.

The most common variety of retro-œsophageal abscess is that due to Pott's disease of the lower cervical or upper dorsal region. The symptoms are obscure, and an exact diagnosis is not often made during life. Death may occur quite suddenly where the previous symptoms have been so slight as to be easily overlooked. The following is a fair example:

A girl two years old was admitted to the Babies' Hospital with caries of the upper dorsal region of two months' duration. The patient was kept in bed and a plaster-of-Paris jacket applied. About a month later dyspnœa was first observed; this was at times quite intense, and again almost absent. It was always on inspiration, expiration being easy. No explanation for this was found in the lungs. There was no difficulty in swallowing, and very little cough. After these symptoms had lasted for about a week, the child while eating was suddenly seized with violent dyspnœa, and in a few moments became completely asphyxiated. Tracheotomy was immediately done, and by means of artificial respiration the patient was restored to comparative comfort. About two hours later a second attack occurred, and the patient died in an hour. At the autopsy there was found an abscess a little larger than a hen's egg, containing about two ounces of curdy pus, overlying the bodies of the first three dorsal vertebræ and communicating with them. These vertebræ were carious. The right pneumogastric nerve, an inch and a half above the bifurcation of the trachea, was compressed between the abscess and a large tuberculous lymph node, with the capsule of which it was blended. In the lungs were a few small tuberculous deposits and the usual conditions found in death by asphyxia. The dyspnœa seems to have been of nervous and not of mechanical origin, and caused by irritation of the

pneumogastric. The fatal issue was apparently from an increase of the pressure upon the nerve.

I have seen but one other case, and this closely resembled the one reported. In the thirteen cases collected by Griffith the symptoms in all were much alike. Dyspnœa, usually of a spasmodic character, was prominent in nearly all, and generally it was the most marked symptom. It was more marked on inspiration, and often accompanied by a spasmodic cough, suggesting laryngeal stenosis. The voice was affected in but two cases, in one complete aphonia being present. It is striking that in no case was there any difficulty in swallowing, in marked contrast to retro-pharyngeal abscess. Swelling in the neck was noted in but three cases. Spinal caries was stated to be present in seven cases and absent in two. The final attack of asphyxia sometimes came without warning, sometimes was preceded for several days or longer by milder attacks.

The diagnosis of this condition is very difficult, and a positive diagnosis almost impossible. It may be suspected in cases of Pott's disease of the lower cervical or upper dorsal regions, when there is spasmodic inspiratory dyspnœa, especially if accompanied by irritative cough. It should, however, be remembered that precisely similar symptoms may depend upon the irritation of a tuberculous node, and that the sudden asphyxia is exactly like that caused by the ulceration of such a node into the trachea or a large bronchus. The latter, however, may occur without the presence of Pott's disease. If the abscess is higher up, there may be a lateral swelling on either side of the neck, just above the clavicle. In most of the cases there are no external signs of disease. Such abscesses are too low to be reached by digital examination of the pharynx. The attack of asphyxia may also be confounded with that due to the presence of a foreign body in the larynx.

The prognosis in cases of retro-œsophageal abscess is exceedingly bad. Death usually results from pressure upon the pneumogastric, as in the cases reported. The abscess may rupture into the œsophagus and recovery follow. This termination is very rare, but such a case has been reported by Knight. A fatal one is reported by Löschner and Lambl. The abscess may burrow along the œsophagus into the abdominal cavity and excite peritonitis; finally, it may open externally.

But little is to be said under the head of Treatment. The symptoms are rarely definite enough to justify a radical surgical operation. Tracheotomy gives but temporary relief to the asphyxia. This operation should be performed, however, in every case, because of the impossibility of making a diagnosis of retro-œsophageal abscess from other conditions in which the operation might be curative.



## CHAPTER V.

*DISEASES OF THE STOMACH.*

It is difficult wholly to separate diseases of the stomach from those of the intestine. Although in older children they are often quite distinct, in infancy they are more frequently associated; but at one time the gastric symptoms may be prominent, and at another the intestinal symptoms. Functional disorders particularly are likely to involve the whole tract. Serious organic lesions are more frequently limited in their extent either to the stomach or to the intestine. The former are rare, while the latter are very common. The diseases in which the stomach is alone or chiefly involved will be considered by themselves. Those in which both the stomach and intestine are involved are classed with the intestinal diseases, as the intestinal symptoms usually predominate.

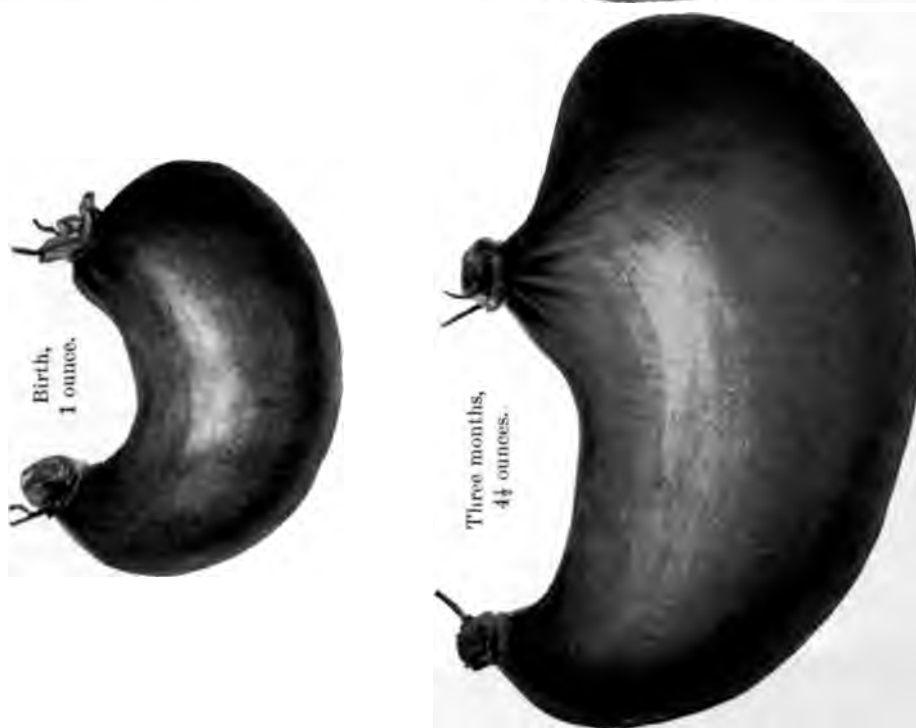
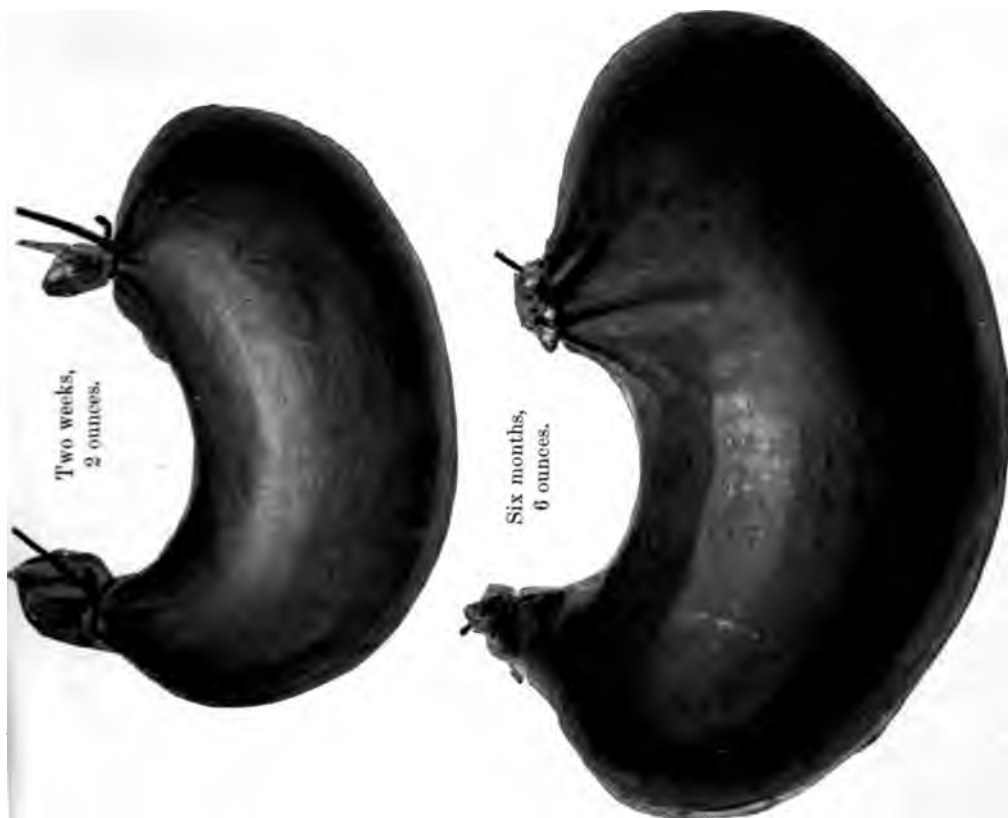
## DIGESTION IN INFANCY.

The first step in the process of digestion in the newly-born infant is sucking. During this act the nipple is grasped between the lower lip and tongue below, and the upper lip and jaw above. The back of the mouth is closed by the palate. A strong downward movement of the lower jaw causes a partial vacuum in the mouth, and produces the suction force which causes the milk to flow. Sucking can be carried on only when the nose is free for respiration and the palate and upper jaw intact. Children with deformities of the mouth, like cleft palate and harelip, suck only with the greatest difficulty, and complete nasal obstruction prevents nursing.

**The Saliva.**—This is present at birth only in very small amount, and the part which it plays in digestion in early infancy is an insignificant one. During the third and fourth months it increases markedly in quantity, and at this time it possesses quite actively the power of transforming starch into sugar. This property is present only to a very slight degree during the early weeks.

**The Stomach.** The position of the stomach in the fetus is nearly vertical. In the newly-born child it lies somewhat obliquely in the abdomen, and at the end of infancy has almost reached the transverse position. The stomach at birth is nearly cylindrical, but the fundus increases in size rapidly during the first year, although it does not reach its full development until early in childhood. In *PLATE VII* are shown the actual size and shape of the stomach at various periods. In the following table are given the results of most numerous measurements of the stomach, which I have personally made in many young infants under fourteen months of age:

PLATE VII.





*The Capacity of the Stomach.*

| AGE.          | Number of cases. | Average capacity. | AGE.              | Number of cases. | Average capacity. |
|---------------|------------------|-------------------|-------------------|------------------|-------------------|
| Birth .....   | 5                | 1.20 oz.          | 12 weeks .....    | 6                | 4.50 oz.          |
| 2 weeks ..... | 7                | 1.50 "            | 14 to 18 weeks .. | 12               | 5.00 "            |
| 4 " .....     | 4                | 2.00 "            | 5 to 6 months ..  | 14               | 5.75 "            |
| 6 " .....     | 11               | 2.27 "            | 7 to 8 " .....    | 9                | 6.88 "            |
| 8 " .....     | 4                | 3.37 "            | 10 to 11 " .....  | 7                | 8.14 "            |
| 10 " .....    | 2                | 4.25 "            | 12 to 14 " .....  | 10               | 8.90 "            |

In brief, the average capacity was, at birth, one and one-fifth ounces; at three months, four and a half ounces; at six months, six ounces; at twelve months, nine ounces.

*Gastric Digestion.*—The part taken by the stomach in digestion is not so important in infants as in adults. The function of the stomach is largely that of a reservoir, into which the milk is received and from which it is allowed to pass gradually into the intestine; the gastric process is only a preliminary and partial one, even in the digestion of protein, this being completed in the intestine.

The gastric juice acts chiefly upon the protein of the food; the digestive agents being pepsin and hydrochloric acid. It is pretty well established that protein digestion in the stomach does not go beyond the stage of peptone formation. The amount of gastric juice secreted is very large. In experiments upon animals it has been found to be nearly as great as the volume of milk taken.

Pepsin is found in the stomach at birth, and even in the foetus as early as the fourth month. In fifteen minutes after feeding the reaction of the stomach contents is always acid. Free hydrochloric acid can not usually be demonstrated until about an hour after feeding, then only in small quantities, and in very many cases not at all. The reason for this is, that the acid combines with the casein and the salts of milk, those of cow's milk in particular having a great power of combining with hydrochloric acid.

The coagulation of milk in the stomach is accomplished through the agency of the rennet ferment. Many good authorities consider that this is not a separate substance, but that coagulation is one of the properties of pepsin. Coagulation is the first change which the milk undergoes in the stomach. Woman's milk coagulates in loose flocculi and quite imperfectly. Cow's milk, unless diluted, coagulates in firmer, rather compact masses. Under the influence of pepsin and hydrochloric acid, solution of this coagulum now begins; but this is only partially accomplished in the stomach. It goes forward much more rapidly in the case of woman's milk, because the amount of casein is less and because of the smaller curds. The fluid portion of the milk begins to leave the stomach very soon after the meal, and even during the first half hour a considerable

part passes into the intestine. At the end of an hour the stomach in a young infant is often empty. If the food is cow's milk, not only are the coagula firmer, but the amount of casein present is much larger, and hence the milk is retained in the stomach a considerably longer time; even then some of it passes but little changed into the intestine. The existence of a fat-splitting ferment in the stomach of infants is now generally admitted, though it plays but a small rôle in digestion.

The duration of gastric digestion varies with the age of the infant and with the food. During the first month the stomach of healthy nursing infants is usually found empty in an hour and a half after feeding, often in one hour. In those taking cow's milk the average is at least one hour longer. In infants from two to eight months old the average is two hours for those receiving breast-milk, and two and a half to three hours for those fed upon cow's milk. The time is influenced by the size of the meal taken and by the composition of the food. The water and milk sugar first pass into the intestine, then the protein in various stages of digestion, and, lastly, the fat. The higher the proportion of fat in the meal the longer the food is retained in the stomach, and also the smaller the amount of gastric juice secreted. Very little absorption takes place from the stomach. There is here absorbed a certain proportion of the sugar and salts, and a small amount of the nitrogenous material, but practically no water or fat.

The bacteria of the stomach are very few as compared with those of the intestine, and no varieties are constantly present.

**The Intestines.**—The length of the small intestine at birth is about nine feet; that of the large intestine about eighteen inches. The great length of the sigmoid flexure is the most striking peculiarity, this being nearly one-half the length of the large intestine.

*Intestinal Digestion.*—All the important elements of food—protein, carbohydrates, and fat—are acted upon by the pancreatic juice. The protein is converted into peptones by trypsin. How much of the protein of the milk is left for intestinal digestion, depends upon how well the stomach has done its part. In every case something is left; in most cases a large part of the protein passes but little changed into the intestine. The digestion of protein is completed by the erepsin of the intestinal juice, which converts peptones and albumoses into amino acids. In this form the nitrogenous portion of the food is finally absorbed.

The amylolytic ferment of the pancreas has the power of converting starch into maltose. This action is feeble during the first five or six months, but is present even in early infancy. Milk sugar is changed into galactose and glucose; and cane sugar and maltose into glucose through the agency of the intestinal and pancreatic juices. Fats are partly emulsified and partly saponified by the pancreatic juice in connection with the bile.

**Absorption.**—From the small intestine absorption takes place very rapidly. The protein is absorbed in the form of peptids and amino acids. Sugars of all varieties are changed to glucose during absorption. Fat is absorbed in the form of fatty acids and soaps; but in their passage through the wall of the intestine the fatty acids are converted into neutral fats. Absorption from the large intestine, except of water, is quite imperfect. Fat absorption is very slight. Sugar, salts, and peptones, however, may be absorbed with moderate facility.

**Intestinal Bacteria.**—For the fundamental work upon this subject we are indebted to the researches of Escherich. Bacteria are absent from the entire gastro-enteric tract at birth. They quickly enter by the mouth and rectum, and by the end of twenty-four hours they are usually found in all parts of the intestinal tract. The meconium-bacteria are derived from the inspired air, and hence vary somewhat with surroundings. As soon as the ingestion of milk begins these varieties are displaced, and throughout the period in which the infant has this food exclusively, there have been found in healthy conditions but few varieties which are constantly present. These are the *b. lactis aerogenes*, the *b. coli communis*, and the *b. bifidus*. The number of bacteria vary in different parts of the intestine. They are found in greatest numbers in the cæcum and colon, and are relatively few in the small intestine. The *b. lactis aerogenes* is found most abundantly in the upper part of the small intestine, in small numbers only in the colon, and usually there are none in the fæces.

The *b. coli communis* is found in but small numbers in the upper small intestine, becoming more abundant lower down. In the colon and in the fæces it is present in considerable numbers. The most abundant organism in the large intestine, however, is the *b. bifidus*. A change from a milk diet to a mixed diet of meat and farinaceous food produces a marked change in the character of the intestinal bacteria.

**Fæces.**—The first discharges after birth are called meconium; this is of a dark brownish-green colour, semi-solid, and usually passed from four to six times daily during the first two or three days. On the third day the stools begin to change in character, and by the fourth or fifth day they have usually assumed the appearance of healthy milk-fæces. Under many abnormal conditions the stools may continue to have the character of meconium for a week or more. Meconium is composed of intestinal mucus, bile, the vernix caseosa, epithelial cells from the epidermis, hairs, fat-globules, and cholesterin crystals. For its formation there are necessary the secretions of the intestine and the liver and the swallowing of a considerable amount of amniotic fluid.

**Milk-fæces.**—The normal amount of fæces discharged daily by a healthy nursing infant is from two to three ounces. Such stools have the colour of the yolk of egg. They are smooth, homogeneous, of a soft, but-

ter-like consistency, with an acid reaction, and a slightly acid but not unpleasant odour. The reaction is due to the presence of fatty acids or lactic acid. The colour depends upon bilirubin. The stools of an infant fed upon cow's milk may, in conditions of perfect digestion, differ in no respect from those just described; usually, however, they are firmer, of a paler yellow colour, and may be neutral or even alkaline in reaction. The normal stool contains about 85 per cent of water and 15 per cent of solids, of which the most important ingredient is fat.

The only gases present are hydrogen and carbon dioxide. Sulphuretted hydrogen and marsh gas, to which the odour of adult stools is largely due, are not present.

The protein of both woman's and cow's milk is almost entirely absorbed. The nitrogenous content of the normal stool is derived chiefly from the intestinal secretions and the bodies of the bacteria.

Fat is always present, and forms from ten to thirty per cent of the dry residue of milk-fæces. It is present as neutral fat, fatty acids, and soaps. Sugar is not found, but its derivative, lactic acid, may be present in a small amount. Inorganic salts form about ten per cent of the dry residue. They are chiefly the salts of calcium. Of the biliary elements there are hydrobilirubin, unchanged bilirubin, and cholesterin in considerable amount. The presence of biliary acids is doubtful. Mucus is always present in considerable quantity.

Microscopically there are seen epithelial cells, chiefly of the columnar variety, a few round cells, mucous corpuscles, fat globules and crystals of fatty acids, cholesterin, mucin, crystalline inorganic salts, sometimes bilirubin in crystals, yeast fungi, and bacteria in immense numbers.

If the infant is taking a food containing starch, this may appear to a greater or less extent in the stools, a larger amount in the case of very young infants.

The number of stools during the first two weeks is from three to six daily. After the first month two stools a day are the average; many infants have three, many others but one.

As soon as an infant is put upon a mixed diet, the peculiar characters of the stools disappear, and they come to resemble more closely those of the adult, though remaining softer throughout infancy. They become darker in colour and assume the adult odour, while retaining their acid reaction. The bacteria, while still in great numbers, are more varied than are met with in milk-fæces.

#### MALPOSITIONS AND MALFORMATIONS OF THE STOMACH.

The stomach is sometimes in the thoracic cavity in cases of diaphragmatic hernia. It may be found in a vertical (fœtal) position, variously adherent to the colon and small intestine. Malformations are much less

frequent than those of other parts of the alimentary tract. There may be atresia or stenosis at either orifice, and very rarely a constriction is found near the middle of the organ, dividing it into compartments. The symptoms of atresia at either orifice are persistent regurgitation or vomiting, and death in a few days from inanition.

#### HYPERTROPHIC STENOSIS OF THE PYLORUS.

This condition known also as *pylorospasm* and as *congenital stenosis of the pylorus*, or simply as *pyloric stenosis of infancy*, is not an uncommon one. It is met with in early infancy and is characterised by persistent vomiting, constipation, wasting, marked visible gastric peristalsis, and often a palpable tumour. It is a serious condition, nearly one-half of the cases ending fatally. Little is known of its etiology. Fully four-fifths of the cases occur in males. It has no relation to the method of feeding; a large proportion of the recorded cases have been in nursing infants. The variety of names reflects the different theories which have been advanced to explain its occurrence. By some the condition is considered a primary hypertrophy with a secondary spasmodic element added; by others, as a purely spasmodic condition from gastric or duodenal irritation, possibly due to increased acidity; by still others the spasmodic condition is regarded as primary, with hypertrophy developing secondarily. Pylorospasm has its analogue in other spasmodic conditions of the circular muscle fibres in early infancy. As examples may be mentioned: constipation due to a spastic condition of the sphincter ani, intussusception due to irregular or intermittent muscular spasm of the intestines, and various spasmodic affections of the larynx and bronchi.

The post-mortem findings are remarkably uniform. The pylorus appears as a hard, whitish tumour about the size of a peanut, of almost cartilaginous consistency. Its lumen may be so narrowed as barely to admit a fine probe, while the normal pylorus will usually admit a No. 21 sound, French scale. Frequently water can not be forced through the stenosis owing probably to the fact that the mucous membrane is thrown into folds. The walls of the stomach are often hypertrophied, especially toward the pyloric end. The stomach is usually much dilated; its lower border may be below the navel. There may even be some dilatation of the œsophagus. On section the thickening of the pylorus is seen to be chiefly of the circular muscle fibres. This coat appears to be two or three times the normal thickness. The other coats—submucous, mucous and longitudinal muscular—are thickened but to a much less degree.

**Symptoms.**—The general clinical picture is a striking one. An infant who for the first two or three weeks has shown no signs of gastric disorder, and often has been nursing and gaining regularly in weight, be-



gins to vomit; at first occasionally, but soon habitually. The change from the usual type of vomiting to the forcible and constant vomiting is often abrupt and without evident cause. The vomiting is not the ordinary gastric regurgitation of indigestion but is forcible and projectile. Changes in diet have but a temporary effect upon it, or none at all. The bowels are constipated. The infant wastes steadily, the scales often showing a loss of one or two ounces a day. There is no fever and little or no evidence of pain. There is progressive failure in nutrition and death may occur from exhaustion in from four to six weeks from the beginning of marked symptoms.

*Time of Beginning of Symptoms.*—Exceptionally this is in the first week or even in the first days of life. The average time, however, is after the first week and during the first month, very rarely as late as the sixth or seventh week.

*Vomiting.*—The manner of vomiting is characteristic. It is more forcible than that seen under any other condition. I have often seen an infant fairly shoot out the contents of the stomach to a distance of four or five feet. Food frequently comes through the nose. The vomiting has usually a relation to the taking of food. It most frequently comes directly after the meal, often while the child is still at the breast. After an attack of vomiting, nursing is sometimes resumed with avidity, showing a distinct absence of the usual symptoms of gastric indigestion. All the food is generally expelled at one time. The frequent regurgitation of small amounts is seldom seen. Generally vomiting does not occur at night unless the child is nursed at that time. The vomited matters at first consist only of food, often but little changed. After a time there is mucus, sometimes in large quantities. The amount vomited at one time is often considerably greater than the meal just taken, indicating a considerable retention of food in the stomach. Some of these children vomit regularly after every feeding; others retain two or three feedings and then expel the whole amount. The frequency of vomiting varies from once or twice to six or eight times a day. Owing to the loss of fluid by vomiting the urine is usually very scanty. There is no uniform change in the gastric secretions, but there is frequently hyperacidity present.

*Bowels.*—Obstinate constipation is the rule. If the pyloric obstruction is complete the stools resemble meconium. Exceptionally diarrhœa is present. I have seen it in but a single case and here the obstruction was not complete.

*Wasting.*—Progressive wasting is one of the striking symptoms, and a close observation of the weight one of our best guides to the progress of the case. If the loss is only two or three ounces a week the outlook is hopeful; while if this amounts to two or three ounces a day the condition should be considered most critical. The rate of the loss depends natur-

ally upon the completeness of the obstruction and it is proportionate to the amount of vomiting and the degree of constipation.

*General Appearance.*—At first nothing abnormal is seen, but soon all the evidences of rapid malnutrition are present, without, however, the other usual symptoms of indigestion, such as might be expected with the vomiting. The tongue is usually clean; the appetite often voracious; there are no eructations of gas; the breath is sweet.

*Peristalsis.*—On examination of the abdomen the epigastrium is usually full and the lower half of the abdomen sunken. If the skin is



FIG. 49.—GASTRIC PERISTALSIS IN PYLORIC STENOSIS. (Thomson).  
Patient eight weeks old.

bared and the patient placed in a good light the characteristic peristaltic waves are seen which are the most diagnostic feature of the disease. One should not expect to see them if the stomach is empty; they are best seen immediately after taking food or water. When not appearing spontaneously they may often be excited by slight friction or tapping of the epigastrium. There is seen a slowly moving wave from left to right. First a ball-like tumour appears just below the ribs on the left side (see Fig. 49). It is usually about one and a half to two inches in diameter and slowly moves toward the right and slightly upward. It disappears just beyond the median line. It is repeated every minute or two. Sometimes one wave is quickly followed by another. These gastric contractions can hardly be mistaken for anything else. They may be accompanied by slight evidences of pain.

*Tumour.*—The hardened pylorus can with careful attention to details be felt in most cases. It may be obscured by distention of the stomach or the colon or by enlargement of the liver. The pylorus may be displaced. The position of the tumour is therefore of less importance in diagnosis than its character. It is usually felt about one and a half to two inches below the free border of the ribs, just inside of the right

mammary line. It is felt only during contraction of the stomach, i. e., best during active peristalsis. It appears somewhat smaller than the little finger and about three-fourths of an inch long, somewhat like a small spool.

*Course of the Disease.*—Two types of cases are seen: (1) the acute, the usual type which, unless relieved by medical or surgical treatment, generally proves fatal in one or two months; less frequently, and when the symptoms are of a milder type, after persisting for several weeks or months, the vomiting gradually subsides and the patient recovers; (2) the subacute or chronic form, which is very rare, but which may give symptoms at irregular intervals during infancy and early childhood. The acute cases differ much in severity but little in other respects. The chronic cases may show periods of exacerbation for years. These exacerbations are sometimes apparently excited by attacks of indigestion. In this type correct diagnosis is seldom made unless operation is done or the case comes to autopsy.

**Diagnosis.**—The diagnosis of pyloric stenosis of infancy is usually easy after two or three days of observation, but may be impossible at the first examination, owing to the difficulty of obtaining the most distinctive signs—the peristaltic waves and the tumour. The time of onset and nature of the vomiting are very suggestive, but not quite conclusive. It has been mistaken for cerebral disease on account of the projectile vomiting and obstinate constipation. In the rare cases seen in older children it might be confounded with cyclic vomiting. However, the query arises whether some of the cases diagnosticated cyclic vomiting may not be of this kind. I have myself seen one such. Usually, however, the only difficulty is to distinguish between the vomiting of gastric indigestion and that of pyloric stenosis. The occurrence of vomiting in nursing infants who have previously thriven on the same food, the abruptness of the development of the vomiting without assignable cause, and its persistence in spite of all treatment, should set one right. Cases in which there is atresia of the duodenum or other part of the small intestine may be mistaken for pyloric stenosis in which the symptoms begin soon after birth. However, in atresia all the symptoms are altogether more severe and the condition is usually fatal in a few days. I have seen one case of partial obstruction of the duodenum due to pressure by a band in which persistent and projectile vomiting and gastric peristalsis were present. The vomited matters, however, were green from the presence of bile. This does not occur in pyloric stenosis.

**Prognosis.**—The condition is always serious, and even with the most approved methods of treatment the mortality is large. I believe that fully fifty per cent of the cases prove fatal. Much, of course, depends upon early diagnosis and proper treatment. Some writers who include in the group of pyloric stenosis many cases regarded by them as milder

types of the disease, give, of course, a much lower mortality. The tighter the obstruction—as indicated by persistence of vomiting in spite of stomach washing, stools of a meconium character, and rapid wasting—the worse the prognosis.

**Treatment.**—Some surgeons argue that, given a correct diagnosis, the only rational treatment is operation, all other measures being only a waste of time and lessening the chances of surgical success because of the weakened condition of the patient. On the other hand, so high is the mortality after surgical operation and so great are the difficulties of after-treatment, even when the immediate result of the operation is favourable, and so many are the undoubted cases which have recovered without operation, that most physicians favour a faithful and patient trial of other measures before referring the case to the surgeon, and recommend operation only as a last resort. One's view of treatment will naturally be modified according to the etiological factor he holds to be most important. If the obstruction is chiefly from tonic spasm, there is no reason why this may not relax and complete recovery take place. If the obstruction is chiefly due to congenital hypertrophy with only a moderate amount of spasm, and this secondary, little that is permanent is to be expected by medical means alone. It is my own belief that both of these types of cases are seen: the one in which the obstruction is nearly all due to spasm, and the other in which the hypertrophy is the more important factor. It is certain that many cases have recovered completely and permanently without surgical aid. A considerable number have come under my own observation. We should, therefore, I think, approach these cases with the knowledge that the condition is a serious one, that the chances of the patient's recovery are only about even under any method of treatment, that there is a fair prospect of cure by medical measures alone, but, finally, that some cases can be saved only by operation.

**Medical Treatment.**—This consists in diet and stomach washing. If a child is nursing and the milk is normal, weaning is not generally advisable. Small meals, not too near together, are essential. The breast should be given at three-hour intervals, and the nursing period varied from three to eight minutes, according to the amount obtained. It is often advantageous to pump the breasts and give a definitely measured amount of breast-milk. Usually for a child a month old not more than two ounces should be allowed at one feeding. On no account should an infant be weaned immediately because of the development of the symptoms of pyloric stenosis. For some infants who have been artificially fed nothing succeeds as well as a wet-nurse. The chief objection to the breast-milk is its high fat which sometimes increases the vomiting.

For infants who are artificially fed a few general principles are pretty well established. In all milk formulas the fat should be low,

usually less than that in whole milk. The formulas from skimmed milk have usually, in my experience, succeeded best. The addition of fat in the form of olive oil can often be made when the fat of milk is not tolerated. Other things besides milk which are sometimes useful are, egg albumin and beef juice. Feeding should be regular and not oftener than every three hours, and the amount at one time from one and a half to three ounces.

No one thing is better attested than the beneficial effects of stomach washing. It empties the organ of food and mucus, and it certainly aids in allaying spasm. I prefer the use of water at 108° to 110° F., rendered alkaline by the addition of one per cent of bicarbonate of soda. It is desirable to see how much food there has been retained in the stomach; a measured amount of water should therefore be introduced and then removed. The washing should be done about two and a half hours after feeding, and repeated twice in twenty-four hours. It should be continued for a considerable period. In cases which recover it has often been found necessary for six to eight weeks twice daily, and for three or four months once daily. Hot applications over the epigastrium may possibly aid in relaxing spasm, but are of much less value than stomach washing. The administration of drugs, especially preparations of opium and belladonna, for the same purpose, is advocated by many, but in my experience they have been entirely without value. The usual effect of stomach washing and changes in diet are a cessation of, or at least a great diminution in, the vomiting. But it should not be discontinued because of this improvement. The loss of weight is less rapid, then ceases, and afterward a slow gain occurs; but the condition of the patient continues critical for some months.

*Indications for Operative Interference.*—In other cases no improvement whatever results from medical treatment; the vomiting is as frequent and as severe as ever; the daily loss in weight may be as much as two ounces; and the stools indicate that nothing passes the pylorus. If such conditions have been observed to exist for several days, to postpone surgical interference is useless. The surgical aspects of these cases are fully treated in works on surgery. The operations chiefly done are gastro-enterostomy and divulsion (Loreta's operation). Each has its advocates. The weight of opinion seems now in favour of the former operation. The immediate dangers are considerable. Shock is generally marked in these little patients, but in my own experience less than was expected. Some of these wasted infants of seven or eight pounds have gone through an operation which consumed thirty-five minutes in a manner most surprising. Hemorrhages and peritonitis are also risks to be reckoned with. The after-treatment is most important, and even after a successful operation the dangers are by no means passed, the child's life often hangs by a thread for two weeks or more. Exhaust-

tion from shock and feeble assimilation, inanition from a continuance of the vomiting or the development of diarrhoea, both common symptoms, may carry off the patient. The post-operative treatment should be in the hands of the physician rather than the surgeon. To supply fluid immediately after operation, nothing is better than the continuous introduction of water into the bowel by the "Murphy," or "drop method." After operation vomiting may sometimes be allayed by placing the child in a semi-erect position. Feeding should be begun after twenty-four hours with breast-milk if possible, at first in teaspoonful doses, the amount being gradually increased according to the child's symptoms. The nutrition for the first weeks is nearly always a matter of much difficulty and taxes the resources of the physician to the utmost. If breast-milk can not be obtained, cow's milk should be given, modified according to the child's symptoms, preferably with a rather low fat percentage. To keep the child perfectly quiet after feeding is very necessary for a long time. Relapses occur in a very small proportion of the cases treated by forcible stretching, and I have seen a relapse in a case treated by stomach washing and diet, but it is not a common occurrence.

#### VOMITING.

Vomiting is one of the most frequent symptoms of disease in infants and young children, and occurs from a wide variety of causes. The physician must have in mind both its common and its uncommon causes. Vomiting takes place with great facility in young infants even from slight causes, owing to the position and shape of the stomach.

1. *Vomiting from Overfilling of the Stomach.*—This is often seen in nursing infants, and there may be no other symptom of disease. It comes within a few minutes after nursing, is easy and without effort, and the food is but little changed. It may be excited by moving the child or making undue pressure upon the stomach. It often comes with eructations of gas or air which has been swallowed.

Vomiting from overdistention may be regarded as a safety-valve, and requires no treatment except to diminish the quantity of food.

2. Vomiting is almost invariably present in cases of *acute gastric indigestion and acute gastritis*. With the former it does not usually come immediately after feeding, and it may be delayed for several hours; with the latter it is usually persistent. The vomited matter consists of the contents of the stomach, but often mucus, and, in severe cases, bile and traces of blood may be vomited for some time afterward.

3. In the *hypertrophic stenosis of the pylorus* of early infancy, uncontrollable vomiting without fever is the principal symptom. (See previous Chapter.)

4. In *acute intestinal obstruction* vomiting is rarely absent, and in

most cases it is persistent. In the newly born, persistent vomiting is almost invariably dependent upon congenital obstruction of the intestine, which is most frequently in the duodenum. In malformations of the colon and rectum it is less constant and appears later. In intussusception, vomiting is forcible, immediately excited by the taking of food, and is at first bilious, but later may become faecal.

5. Vomiting is a frequent and almost a constant symptom of acute peritonitis, whether localised or general, of which appendicitis is the usual cause. It is then associated with abdominal distention, tenderness, and fever.

6. In certain *nervous diseases*, especially tumour of the brain and acute meningitis, whether cerebro-spinal or tuberculous, vomiting is very common. Cerebral vomiting is usually forcible or projectile. It may have no relation to meals. Headache, dulness, slight fever, constipation, and irregular pulse and respiration are usually present sooner or later.

7. In infants, and less frequently in older children, vomiting is one of the most frequent symptoms to mark the *onset of acute febrile diseases*, especially the beginning of scarlet fever, pneumonia, and malaria.

8. An accumulation in the blood of various *toxic* materials may provoke vomiting; the best known example is uræmia. In cyclic vomiting it is quite probable that the cause is the accumulation of some toxic substance in the blood. The absorption of poisons taken in with milk or other food, or developing in the gastro-enteric tract, may excite vomiting. In some of these conditions it is possible that the vomiting may be eliminative. The cases dependent upon renal disease are discovered by examination of the urine. The other forms are often exceedingly obscure, and recognised only by the exclusion of all other causes of vomiting.

9. Vomiting may be *reflex* from irritation in the pharynx. This is frequent in young infants, who may induce vomiting by stuffing the fingers into the mouth. In certain cases the irritation from worms in the intestinal tract may cause vomiting, and it is possible that even dentition may produce it.

10. *Habit* is a frequent cause, in cases of chronic vomiting. I have seen many children who had the power of vomiting at will anything in the nature of food which they did not like, yet who would retain other food with no difficulty. One such child would tolerate large doses of quinine, to which he had no aversion, without the slightest disturbance. In young infants a habit of regurgitating the food may be acquired, so that this takes place more or less during the process of digestion after every meal. This is sometimes preceded by a movement of the mouth and fauces resembling swallowing, until finally the milk appears in the mouth. Habit is a potent cause in continuing vomiting where it has occurred frequently. In children who have this habit the most trivial

cause will provoke it. It may be present without any other sign of gastric disease, and appears simply to depend upon exaggerated reflex irritability of the organ. I have seen a number of children who up to the third or fourth year objected so strenuously to taking solid food that they would immediately vomit it, no matter of what variety or in how small a quantity, although fluids were taken and easily digested.

11. *Chronic vomiting* may depend upon habit, as just described, or upon chronic indigestion; or it may be associated with chronic pulmonary disease—vomiting here being excited by the attacks of cough, at first only when the paroxysms are severe, and later even when they are slight.

The diagnosis of a case in which vomiting is the chief symptom may be difficult. The first important distinction to be made is between cases in which the vomiting is of gastric origin, and those in which it depends upon other causes. It is only by a careful consideration of the associated symptoms that an accurate diagnosis can be reached.

The treatment of vomiting is the treatment of the cause upon which it depends.

#### CYCLIC VOMITING.

This is quite a frequent condition; it has, however, attracted but little attention except in this country. Although the clinical picture is a very clear and definite one, its exact pathology is undetermined. It has also been described under the names *periodical vomiting* and *recurrent vomiting*. It is characterised by periodical attacks of vomiting, which recur at regular or irregular intervals of weeks or months, apparently without any adequate exciting cause. The usual duration of the attacks is two or three days, during which all attempts to control the vomiting are usually without avail, but at the end of this time it generally ceases spontaneously.

**Etiology.**—The first attacks are usually seen between the ages of two and four years, but they may date back to infancy. The two sexes seem to be almost equally liable. A few of the patients are strong children, but the great majority are rather delicate and of a highly nervous temperament. The cases are seen chiefly in private practice, often occurring among those who have the best surroundings. In most cases the antecedents of patients are of a neurotic type. The attacks are not usually traceable to distinct or flagrant errors in diet, and yet the habitual diet seems to bear some relation to the disease. The exciting cause is often a nervous one—great fatigue or unusual excitement, sometimes a railroad journey or a child's party; in many instances it seems to be induced by some minor illness having no relation to the digestive tract, such as an attack of tonsillitis or bronchitis. In children subject to this condition serious diseases, such as scarlet fever or measles, may be ushered



in by prolonged and repeated vomiting, which usually ceases before the end of the febrile period. General anæsthesia, especially by ether, is very likely to precipitate an attack.

**Symptoms.**—The clinical picture presented by these cases is very characteristic, and is well illustrated by the history of the following case:

The patient was a well-nourished boy of six years when he first came under treatment. He belonged to a neurotic family, and the attacks dated back to infancy. From this time they had recurred usually at intervals of a few months; occasionally five or six months would pass without one. The symptoms in all the attacks were similar in kind, differing only in degree. They were preceded by a prodromal period lasting from twelve to twenty-four hours, marked by languor, dulness, dark rings under the eyes, loss of appetite, and a general sense of discomfort in the epigastrium. At this time the temperature was generally slightly elevated. The vomiting then began suddenly. It was attended with great retching and distress; it was often repeated every half-hour or hour for two days. On one occasion it occurred seventeen times in a single night. Vomiting was immediately excited by the taking of any food or drink, but it occurred when nothing was taken. The vomited matters consisted of frothy mucus and serum, frequently streaked with blood, apparently from the violence of the emesis, and often containing bile. The temperature usually fell to about 100° F. when the vomiting began, and continued at or below this point throughout the attack. By the end of the second day the exhaustion was very marked—so severe, in fact, as apparently to threaten life.

The child lay in a semi-stupor, with eyes half open, lips and tongue dry, rousing at times to beg for water. The pulse was rapid and weak, and sometimes slightly irregular. There was no distention of the abdomen; it was usually flattened. By the third day the vomiting became less frequent and then ceased entirely. Convalescence was rapid, and by the end of the week the boy was almost as well as usual. The attacks continued to recur at gradually lengthening intervals until they finally ceased altogether at about the twelfth year.

A great number of these cases have come under my observation, and in many patients I have had an opportunity to witness several attacks. The usual duration is one to three days. In one child they lasted regularly for five days. Occasionally a severe attack will last a week. The average number of attacks is three or four a year.

Prodromal symptoms are present in most of them—headache, general languor, coated tongue, and anorexia are the most frequent; in some there is marked constipation, with a history of very white stools for some time. But it is not uncommon for an attack to occur in the midst of apparently perfect health. The tongue is usually coated at the

beginning of an attack, and at its height it is often dry and brown. The abdomen seems empty and its walls sunken; pain and tenderness are both rare. The bowels are usually constipated and move only with difficulty by artificial means. Very exceptionally there may be diarrhoea with foul stools.

There is, as a rule, no desire for food, but the continual cry is for water to quench the constant, burning thirst. The pulse after the second day becomes rapid, soft, and often somewhat irregular. The respiration is shallow, and at times this also may be irregular. The temperature is usually under 100.5° F., rarely it may be 102° or 103° F. The usual low temperature is a point of much diagnostic value. The patients are dull, apathetic, and usually wish to be left alone. Head-ache is very common.

The disposition to vomit is sometimes so great that patients are afraid to move or even to talk lest it may be provoked. The vomited matter is often large in amount, considering that the patient is fasting. It is essentially gastric juice, containing free HCl, mucus, serum, many epithelial cells, and often traces of blood. Less frequently vomiting may occur only two or three times a day. The urine is concentrated, and frequently contains at the height of the attack a trace of albumin, a few hyaline casts, and some blood cells. An increase in the renal secretion may be the first sign of improvement. There is usually an excess of indican both during and between attacks. A condition practically constant, and first pointed out by Edsall, is the presence in the urine of acetone, diacetic and oxybutyric acids. These substances appear in the urine so early in the attack that they can not be ascribed to starvation, and are therefore of much diagnostic value.

**The Nature of the Attacks.**—These cases have little in common with the ordinary attacks of indigestion. With our present knowledge they are to be regarded as explosions due to faulty metabolism, and there are many reasons for the opinion that the vomiting is an effort at elimination. It is probable that not all the cases depend upon the same condition.

**Prognosis.**—Although these patients very often seem to be most alarmingly ill, the danger to life is slight. I have seen but one fatal case, and in this the diagnosis is open to question, as no autopsy could be obtained. Griffith reports two fatal cases, the autopsy in one showing nothing characteristic. The probabilities are always in favour of a recurrence of the attacks. In most of the patients who have been observed they have extended over a series of several years, although by a careful régime much may be done to reduce their frequency and diminish their severity. In a small proportion of cases they may be stopped altogether. Toward puberty there appears to be a strong tendency to spontaneous recovery.

**Diagnosis.**—Organic disease of the brain and kidneys must first be excluded. The first attacks witnessed may strongly suggest the onset of tuberculous meningitis; and only the course of the symptoms may show that this is not present. Usually a history of many previous attacks may be obtained. From acute indigestion, cyclic vomiting is differentiated by the fact that the attacks are not brought on by indigestible food, and also by the persistence of the vomiting, and the early presence in the urine of the acetone bodies. It is distinguished from gastritis by its severity, the shorter duration of its symptoms, and its self-limited course.

Appendicitis is excluded by the absence of pain, tenderness, and muscular rigidity; intussusception by the fact that the symptoms are less severe, by the absence of blood and mucus from the stools, and by the fact that intussusception is usually seen in infancy.

**Treatment.**—When the premonitory symptoms appear, starvation and free purgation offer the best prospect of aborting an attack. If the vomiting has once begun, nothing seems to have the slightest influence in controlling it. It is usually increased by the taking of food or drink or by any medication by the mouth, and all should be withheld. The patient should be kept absolutely quiet and water given, per rectum, at regular intervals, usually six to eight ounces, four or five times a day. This keeps up the urinary secretion, allays thirst and often restlessness, and when it is retained usually adds much to the patient's comfort. When the vomiting has ceased for several hours it is not likely to recur if food is very judiciously administered, at first in small quantities. Broth, barley water, kumyss, or small quantities of iced milk and lime-water in equal proportions may then be given.

The alkaline treatment has been strongly advocated; it consists in giving between the attacks bicarbonate of soda in doses of fifteen to thirty grains three times daily, and, when the prodromal signs of an attack appear, to administer very large doses, as much as thirty grains every hour. I have used this plan of treatment with some apparent success and think it deserves a trial. In the interval the treatment should be chiefly dietetic. All sugar and sweets should be carefully excluded. The diet should consist principally of meat, green vegetables, milk, cereals in moderate amount, and stale bread. In addition to careful regulation of the diet the general nutrition should be considered, and the patient's life so regulated that extreme fatigue and exhaustion are prevented. In most cases close attention to these matters has resulted in a very great diminution in the frequency of the attacks.

#### GASTRALGIA.

This term is applied to sudden, severe attacks of abdominal pain. Gastralgia occurs as a symptom in most of the severe attacks of acute

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gastric indigestion; in such cases it is more marked in older children than in infancy. The pain of diaphragmatic pleurisy is often referred to the epigastrium, and may be so severe as to lead one to think that the stomach is the seat of disease. Another cause may be appendicitis. In vertebral caries of the dorsal region epigastric pain is a very frequent, early symptom. It is also common in children who suffer from malaria at the onset of acute attacks, and it may be severe when the febrile symptoms are not well marked. In other cases pain in the stomach is of the nature of a true neuralgia, which may be excited by exposure to cold, by wetting the feet, by drinking ice-water, and by many other causes.

In mild cases there is an intermittent pain, and usually no other symptoms. In severe cases the pain may be so great as to cause pallor, faintness, cold perspiration, and very marked prostration. The epigastrium may be hard and sometimes retracted, the stomach appearing to be in a state of spasm.

The principal interest attaches to diagnosis. If the pain is acute, one should carefully exclude appendicitis, renal and hepatic colic, ulcer with perforation, and all acute inflammatory conditions in the abdomen; if more chronic, Pott's disease should not be forgotten.

During the attacks the patient should be put to bed, and counter-irritation used over the stomach, best by means of a turpentine stupe or a mustard paste. Internally there should be given hot water containing a few drops of brandy or gin and five drops of spirits of chloroform; all food should be withheld. Hot bottles should be applied to the feet if they are cold. In the interval between the attacks the treatment should be directed to the patient's general condition; especially should the cause be discovered, and if possible removed. In cases of recurring pain of a neuralgic character arsenic in the form of Fowler's solution, one or two drops three times a day, may prove of benefit. In all cases attention should be directed to the diet.

#### ACUTE GASTRIC INDIGESTION.

This occurs whenever the stomach is unequal to the task imposed upon it. It may be either because the task is too great or because the capacity of the stomach for work is diminished. Under these two heads we may group the principal causes of acute indigestion.

Under the first head the most important thing is the giving of improper food. In infants this is sometimes improper breast-milk; but more often cow's milk containing too high fat. Other common causes are sudden weaning or any other abrupt change in diet, the too early use of solid food, and overloading the stomach. In older children the usual causes are indigestible articles of food, such as unripe fruits, pastry, imperfectly cooked cereals, etc., overloading the stomach, and

swallowing food without sufficiently masticating it. Conditions which may diminish for the time the capacity of the stomach for work are fatigue, depression induced by atmospheric heat, chilling of the surface, especially the extremities, dentition, and the nervous impression caused by the onset of any acute disease. The effect is seen both on the glandular and muscular apparatus of the stomach. The secretions are diminished or altered in character, and the motility of the organ is arrested.

**Symptoms.**—One of the first consequences of arrested gastric digestion is that the food remains long in the stomach. Instead of the stomach's being empty in about three hours after feeding, as is normal in infancy, the food may remain in it five or six hours, or even longer. The irritation from this undigested and fermenting mass excites vomiting, which usually ceases after the stomach has been emptied. The vomiting may be preceded by nausea, pain, and constitutional depression which varies with the age and susceptibility of the child; in infants it may be very alarming.

The nervous symptoms are sometimes of a striking character. There may be dulness, stupor, and sometimes contracted pupils, so as to suggest opium narcosis, or there may be restlessness, and even convulsions. There is also marked prostration and fever. The temperature in most cases of acute indigestion is from 101° to 103° F.; not infrequently it rises to 104° or 105° F. The tongue is coated and the appetite entirely lost. In infants these symptoms are usually associated with or followed by more or less intestinal disturbance—generally diarrhœa, with undigested food in the stools. Epigastric distention may be present. Usually the vomiting ceases in from six to twelve hours and after the stomach has been thoroughly emptied the temperature falls. Provided rest to the organ can be secured, and the exciting cause is one that can be removed, the patient may be quite well in two or three days. Relapses are, however, easily excited; and in a susceptible patient it is surprising to see how trivial a cause may excite one.

The diagnosis between a simple attack of acute indigestion and one of gastritis can not be made at the outset. The former is much more frequent, and may be quite as severe, but is of shorter duration. The prognosis in these cases is good, except in very young or very delicate infants.

**Treatment.**—The indications are, to empty the stomach as completely as possible and then to secure for it absolute rest. If proper treatment is employed at the outset, the majority of such attacks can be cut short. Nothing is so efficient in infants as stomach-washing. A single washing usually suffices. If for any reason this can not be employed, the child may take from its bottle a large amount of lukewarm water. The free vomiting which this usually provokes may be sufficient

to cleanse the stomach fairly well, but by no means so thoroughly as stomach-washing. Persistent vomiting is sometimes arrested by giving small quantities of hot water.

The subsequent treatment is chiefly dietetic. Everything should be withheld for six to eight hours, when thin barley water or albumin water may be given in small quantities, e. g., half an ounce to one ounce every hour. After twenty-four hours beef juice or broth may be added, but no milk should be given for two or three days. When begun, it should be skimmed and diluted with five or six parts of water. In a nursing child, the breast should be withheld altogether for twenty-four hours, and then nursing allowed for two minutes every three hours, the time of nursing being gradually increased to three, five, and ten minutes as improvement occurs. The great mistake made in these cases is to begin food too soon and to give too much, especially of cow's milk.

Drugs are relatively of little value. If the measures mentioned have been used promptly they will not often be required. In many cases injudicious medication aggravates the symptoms and prolongs the attack. Unless the bowels have acted freely, calomel (gr.  $\frac{1}{8}$  every hour) may be given until this effect is obtained. Where there is continuous vomiting of very acid mucus and serum, alkalies are indicated—lime-water, chalk mixture, or the subcarbonate of bismuth. It is important to keep the child as quiet as possible. Local applications to the epigastrium are very often useful. Either dry heat may be applied by means of a hot-water bag or hot flannels, or more active counter-irritation by mustard. In older children the stomach should be kept entirely at rest for half a day, only carbonated waters or barley water being allowed in small quantities to allay thirst. Later, broth or beef juice may be given, afterward skimmed milk diluted with lime-water. The patient should be kept upon a very low diet for four or five days.

#### ACUTE GASTRITIS.

In comparison with the frequency of inflammatory diseases of the intestine, those of the stomach are rare, particularly so in infancy. Owing largely to the character of its secretion and its contents, the stomach is much more resistant to infection than are the intestines. Gastritis seldom exists alone, but is usually associated with enteritis or colitis.

**Etiology.**—The causes of gastritis are, in the main, those of acute gastric indigestion—improper food or feeding—to which possibly is added infection. Gastritis may also be caused by the introduction of irritants, which may either be swallowed accidentally or given as drugs.

**Lesions.**—The mucous membrane of the stomach may be the seat of acute catarrhal, ulcerative, or membranous inflammation, all forms ex-

cept the catarrhal being rare. There is also seen a mixed form, which from its cause is usually termed "corrosive gastritis."

*Catarrhal Gastritis.*—This is characterised by hyperæmia of the mucous membrane, exudation of cells into the mucosa, a great increase in the secretion of the mucous glands, and changes in the epithelium. About the only change which can be recognised by the naked eye is congestion and swelling of the mucous membrane. These are usually more marked toward the pyloric end and along the greater curvature. There may be small extravasations of blood into the mucosa. The stomach contains undigested food and mucus, which may be thick and tenacious, adhering very closely to the mucous membrane. The mucus may be stained brown from the capillary hæmorrhages. The stomach may be either distended or contracted. Under the microscope the changes are seen to be almost entirely in the mucosa. In some places there is loss of the superficial epithelium, in others only degenerative changes in it are seen. The mucosa is infiltrated with round cells, this process being rarely diffuse, but generally occurring in patches. The blood-vessels are distended and many small extravasations are seen. Sometimes there is a moderate infiltration of the submucosa. Acute catarrhal gastritis alone is rarely severe enough to cause death. It is usually seen in cases which prove fatal from other causes, particularly diseases of the intestine.

*Gastric softening (gastromalacia)* is a condition dependent upon post-mortem changes—probably self-digestion of the stomach. It is found both where gastric symptoms were present and where they were absent. It is situated nearly always in the posterior wall, and usually covers a considerable area, about one-third or one-fourth of this wall. It is recognised by the gelatinous, translucent appearance of the walls of the stomach, which are so softened that the finger may be pushed through them without force, or that sometimes the stomach ruptures while it is being removed. This condition is rarely seen when the stomach is empty. It can scarcely be mistaken for a pathological condition, if its occurrence is borne in mind.

*Ulcerative Gastritis.*—This was met with six times, not including tuberculous cases, in 330 consecutive autopsies upon infants in the Rabies' Hospital. Three of the patients were less than four months old, and all were females. The ulcers varied from one twenty-fifth to one quarter of an inch in diameter, and usually from ten to fifty were present. They seldom extended to the muscular, and never to the peritoneal coat. The lesion was most marked in the posterior wall, toward the pyloric end and along the greater curvature. Evidences of catarrhal inflammation were present in most of the cases, and in four of membranous inflammation. Lesions in some other part of the digestive tract were present in all but one case, in two there was ulcers of the mesogastrium, in three there was ulceration somewhere in the intestines.

**Membranous Gastritis.**—This is even more rare than the varieties previously mentioned. I have met with it but four times in infants. One case was associated with a membranous colitis; a second case with a streptococcus inflammation of the fauces and larynx in an infant but six weeks old. The œsophagus was not involved in this case; and indeed it often escapes. No Klebs-Loeffler bacilli could be found either in cover-slip preparations or by culture.

To the naked eye the membrane appears of a grayish-green colour; it is adherent, but can be detached in quite large patches. Only a portion of the stomach was covered in any of the cases; in two the principal disease was about the pylorus; in another along the greater curvature. The microscopical appearances resemble those of membranous colitis. There is a pseudo-membrane composed of fibrin, granular matter, epithelial cells, and bacteria. The mucosa shows a moderately dense infiltration with round cells, and in places superficial ulceration. There is also infiltration of the submucosa, and in some places even the muscular coat is involved.

Membranous gastritis occurring in patients dying of diphtheria is not common. Councilman, Mallory, and Pearce noted its presence in only five of one hundred and twenty-seven autopsies.

**Corrosive Gastritis (toxic gastritis).**—This form of inflammation is excited by various irritating and caustic substances, which are usually taken by accident, sometimes for the purpose of producing emesis. The most frequent substances are carbolic acid and caustic alkalies.

The lesions in the stomach depend upon the amount of the substance swallowed, the degree of concentration, and whether the stomach was full or empty at the time. Strong caustics, whether acids or alkalies, usually act more deeply and extensively in the pharynx and œsophagus, for, owing to the spasmodic contraction of the muscles of these parts, often but a small amount of the substance reaches the stomach. Concentrated irritant poisons produce in the stomach, especially along the greater curvature, irregular ulcers, which may be so deep as to cause perforation, or they may affect the mucous membrane only. In severe cases death takes place early, often in a few hours. Dark, ragged ulcers are found in the stomach, the surrounding mucous membrane is the seat of intense congestion, and in places there are extravasations of blood. If death is delayed there are evidences of intense inflammation, sometimes with the production of a pseudo-membrane. If the amount of poison is not sufficient to cause death, and if the patient recovers from the resulting gastritis, a cicatricial condition of the stomach results, which later may lead to stenosis of the pylorus or other deformity of the organ.

**Symptoms.**—*Catarrhal gastritis* can not be distinguished at its beginning from an attack of acute indigestion. There are fever, pain, vomiting, thirst, loss of appetite, coated tongue, and prostration. The pres-



ence of inflammatory changes is indicated by the continuance of these symptoms, particularly the pain, vomiting, fever, and thirst. With the pain there may be epigastric tenderness. All food and liquids are immediately rejected, and even when nothing is taken the retching and vomiting may continue, nothing but frothy mucus or serum being brought up, sometimes streaked with blood. The vomited matters are usually very sour; they may be bilious. The temperature is rarely high except at the outset. After the first or second day it usually ranges between 100° and 101.5° F. Thirst is intense, and all liquids are taken with avidity, especially if cold, even though they are immediately vomited. The tongue is thickly coated with a white fur, and the breath may be foul. The constitutional symptoms are generally most severe at the outset. The usual duration of such attacks is from four to seven days, but with improper management, especially injudicious feeding, the disease may be much prolonged. One attack may follow another until a chronic condition is established. In most of the cases there is some disturbance of the intestines, usually a sharp attack of diarrhoea. Sometimes the gastric symptoms subside after a few days and those of the intestines become the predominant ones. The symptoms above given are those in infancy. In older children there is less fever, prostration, and diarrhoea, but pain and vomiting are prominent. The attacks are usually shorter and altogether less severe.

The rare cases of *ulcerative gastritis* have nothing by which they can be distinguished from the form described, except a more prolonged course and a greater liability to hæmorrhage.

*Membranous gastritis* also presents no peculiar symptoms. In fact, in the cases I have personally seen, the gastric symptoms were insignificant, and the condition not suspected during life.

In *corrosive gastritis* the effects of the caustic may be seen in the mouth and pharynx, the mucous membrane being usually of a gray or whitish colour. Pain and a sense of constriction are felt in the œsophagus and stomach, and thirst is great. Vomiting follows almost immediately, and the matters vomited are usually bloody. The subsequent course in most of the cases is the rapid development of collapse, and death in a few hours from shock. The younger the child the sooner does the case terminate. In irritant poisoning not severe enough to produce death, the symptoms of acute gastritis follow, usually accompanied by more or less enteritis owing to the passage of the irritant into the intestine. There is seen a continuance of the vomiting, pain and epigastric distention, and diarrhoea, and from these symptoms death may result in two or three days. It is extremely rare in infancy for the patient to survive both the stage of shock and that of acute inflammation, so that the deformities of the stomach and the chronic conditions mentioned are practically never met with excepting in older children.

**Treatment.**—Cases of acute catarrhal gastritis are to be managed very much like those of acute gastric indigestion. Thirst may be relieved by swallowing bits of ice. Where there is continuous vomiting of acid mucus, relief is sometimes afforded by repeating the stomach-washing once in twelve hours with a one-per-cent solution of bicarbonate of soda, at 110° F. In older children, beneficial results sometimes follow the use of bismuth subcarbonate (gr. x every two hours); but in infants I have seen but little effect from any form of medication, the reliance being upon rest, careful feeding, and stomach-washing.

Cases of corrosive gastritis require special treatment. The first indication is to administer the proper chemical antidote to the substance swallowed, and the next to use bland mucilaginous or oily fluids, such as milk, albumin water, oils in large quantities, etc. Especially should stomach-washing be avoided. Opium is always required, on account of pain, and should be given hypodermically. The general symptoms are to be treated according to the indications of the individual case.

#### CHRONIC GASTRIC INDIGESTION—CHRONIC GASTRITIS—GASTRIC CATARRH.

Although from a pathological point of view these conditions may not be identical, from a clinical standpoint there is no advantage in attempting to separate them. Nothing distinguishes chronic indigestion from chronic gastritis except that in the latter, in addition to continued derangement of function, there is a greater increase in the production of gastric mucus. Chronic indigestion does not long exist without the production of a certain amount of catarrhal inflammation. This condition in the stomach seldom, if ever, exists without more or less involvement of the intestine, and in the majority of cases the intestinal condition is the more important. In some, however, the gastric symptoms predominate, and it is only those which are here considered.

**Etiology.**—Chronic gastric indigestion may follow acute attacks, or it may be chronic from the outset. If the latter, it depends in infancy upon the continued use of improper food or bad methods of feeding. The improper food is very often a modified cow's milk of improper proportions. The most frequent mistake is the use of too high a percentage of fat. Less frequently the cause is the sugar, especially the use of foods containing much cane sugar or maltose. Other factors of importance are overfeeding, too large meals, unsuitable food, especially solid food for infants. The condition generally accompanies dilatation of the stomach. As a consequence of imperfect digestion, fermentation in the residuum takes place, and the irritating products of this fermentation soon cause a catarrhal inflammation with a production of mucus. Chronic gastric indigestion also complicates most of the constitutional diseases of infancy, especially rickets, syphilis, tuberculosis, malnutrition, and

marasmus. It may follow any of the acute infectious diseases. In older children it is due chiefly to the use of improper food, sometimes to the habit of rapid eating and insufficient mastication, the cause of which is very often carious teeth. It is associated with constitutional diseases as in infancy, and may complicate valvular disease of the heart.

**Lesions.**—The changes found in chronic gastritis are usually confined to the mucosa. In the mild form there are degenerative changes of the epithelium of the tubules, with an increased production of mucus; there may be a slight infiltration of the mucosa with round cells. The more severe form, with marked cell infiltration and the production of new connective tissue, is extremely rare. The submucous coat may be thickened and the muscular coat attenuated. The lesion can not be recognised by the naked eye. The stomach is apt to be more or less dilated, and its surface is coated with thick and very adherent mucus. This lesion rarely exists alone, practically never in infancy, but is associated with similar lesions in the intestines, the latter often being more severe.

**Symptoms.**—*In Infants.*—For our knowledge of the conditions existing in the stomach in chronic indigestion we are indebted to the work chiefly of Cassel, Leo, Troitzky, Wohlmann, and Clarke. The results obtained in the examination of stomach contents have not been uniform, and in practice one should not lay much stress upon the absence of the normal secretions. The presence of mucus in the vomited matters or in the washings from the stomach is a constant feature. This greatly interferes with digestion, even though secretions are normal. The reaction of the stomach is almost invariably acid, but the acidity may be due more to the products of fermentation than to hydrochloric acid. The latter is almost invariably diminished in quantity and is sometimes absent. Free hydrochloric acid is very seldom present. The rennet ferment and pepsin are usually present in normal amount. Fermentation takes place in the fats and the carbohydrates. The results of fermentation are the production of lactic, acetic, butyric, and other volatile fatty acids, which are especially irritating to the mucous membrane. There is an increased production of gas. Food remains long in the stomach because of motor inactivity, which is partly the cause and partly the result of the disease. It often continues after all other symptoms have disappeared.

The most important local symptom is vomiting. It may occur soon or long after feeding. It is often accompanied by frequent regurgitation of small amounts of food, which may begin soon after one feeding and continue quite to the time for the next. In nearly all protracted cases the vomited matters contain mucus, and sometimes this is a conspicuous feature. The regurgitation of a sour irritating fluid occurs even when but little food is rejected, and usually accompanies the belching of gas. In infants some of the most striking symptoms are due to the gas. The

stomach may be distended and hard most of the time, and often so much gas is present that infants find difficulty in taking food. Though evidently hungry, they can take so little at a time that an hour or more may be required to take four or five ounces. That the food remains long in the stomach is best demonstrated by stomach-washing. Instead of the stomach's being empty in two and a half or three hours, as it should be, food may be found four or five hours, and in some cases six or eight hours, after feeding. There may be dilatation of the stomach, especially in rachitic children.

The appetite may be abnormally great, or it may be poor. As a rule, children take less food than in health. The tongue is usually coated. The general symptoms are those of malnutrition; there is constant fretfulness and sleep is irregular or disturbed; the weight is stationary, or there is steady loss; there is also anæmia, and the child's development is arrested. There is nearly always some derangement of the bowels, more often constipation than diarrhœa.

There is little tendency to spontaneous improvement or recovery, the prognosis depending almost entirely upon the treatment employed. Unless relieved the condition is apt to continue, until some serious acute disease develops which may be fatal. In young infants, chronic gastric indigestion should not be confounded with hypertrophic stenosis of the pylorus.

*In Older Children.*—The disease is not so common as in infants. In all cases the most constant symptom is vomiting, which may occur regularly after meals, or only in the morning before breakfast. If the latter, the vomited matters consist chiefly of mucus. In addition to these regular attacks there may be the frequent regurgitation of small quantities of food. There are gastric flatulence and pain, due to hyperacidity or to acid fermentation. The appetite is variable—sometimes inordinate, sometimes entirely lost; it may be capricious, there being usually a craving for highly seasoned food. The tongue is constantly furred, and the breath usually disagreeable. These symptoms are seen in all degrees of severity. Intestinal disturbances are not so frequent as in infancy. Constipation is more common than diarrhœa. The general symptoms are those of malnutrition. There are anæmia, wasting, constant fretfulness, disturbed sleep, and various other nervous disorders.

**Prognosis.**—The prognosis depends upon the age of the patient, the duration of the disease, the surroundings, and upon how well treatment can be carried out. In infants under three months the prognosis as to life is doubtful. If children live to the age of four or five months, they usually recover with proper treatment. These patients do much better in private practice than in institutions. The principal danger from this condition consists in the predisposition it gives to acute diarrhœal diseases in summer.

In older children, as in the case of infants, these symptoms may continue indefinitely; there is little tendency to spontaneous recovery, but under favourable circumstances, with constant care, much may be done for all these patients and many of them may be completely cured.

**Treatment.—Infants.**—The general treatment is too apt to be ignored, but it is just as important as measures directed more specifically to the stomach. A large, roomy nursery, and plenty of fresh air by night and by day, are very important; equally necessary are quiet surroundings and freedom from disturbing conditions which sometimes obtain in the nursery; sometimes under the influence of these alone improvement begins. General friction of the body is useful in delicate children with poor circulation. Infants must be properly covered, and it is of the utmost importance that the feet be kept warm. Of the measures directed to the stomach, two are chiefly to be depended upon—stomach-washing and proper feeding.

Stomach-washing is useful, first, in removing the mucus which is abundant in most of these cases; secondly, in cleansing the organ thoroughly at least once a day, this of itself being most important; thirdly, as a stimulant to the gastric secretions, especially hydrochloric acid. Plain boiled water, or a weak alkaline solution—sodium bicarbonate, one drachm to the pint—may be employed. In the early part of the treatment the washing should be done daily; later, every second or third day. The time selected is not very important, but it is better to make this about three hours after feeding.

The question of diet has been quite fully discussed in the chapter on Infant-Feeding, particularly in the pages in which the feeding in difficult cases is considered. If milk is being given, one should first endeavour to determine which of the elements is the chief cause of the trouble. This is most frequently the fat, and next the sugar.

The quantity of food and the frequency of feeding are both matters of importance. With a serious amount of chronic gastric disturbance in infants over three months old the interval between feedings should not be less than three hours; many do better when the interval is four hours. Small meals of a somewhat concentrated food are usually better than large feedings of a very dilute food. Careful study of the individual child is indispensable to success.

Drugs have a very limited application in the treatment of this condition in infants. Generally they are too much used, and too little attention is given to the details of feeding, by which means alone permanent improvement is reached. The continued use of pepsin and other digestive ferments is irrational and without benefit. Hydrochloric acid, however, may at times prove of considerable value, but it must be given in rather large doses, i. e., five to fifteen drops of the dilute acid after each feeding. But for the relief of one condition drugs may be of considerable

**advantage:** wherever the production of gas and constant eructations are prominent symptoms, the benzoate of soda is sometimes useful. It may be given with the feedings in doses of two or three grains.

*Older Children.*—The management of these cases in older children must be conducted along the lines laid down for infants. With them, stomach-washing can not be easily employed, and other means must be used to clear the stomach of mucus. The best is undoubtedly the use of large draughts of water, as hot as can be borne, an hour before eating. From six to eight ounces should be taken, preferably slowly by sipping. To this may be advantageously added, in many cases, fifteen or twenty grains of bicarbonate of soda.

The diet should consist of skimmed milk diluted at least once, kumyss or matzoon, beef juice, rare meat, and a moderate amount of starchy food, preferably dried bread or zwieback. All fruits should be avoided. All pastry, sweets, nuts, and candies should be absolutely prohibited. With improvement in the symptoms green vegetables may be added to the diet, and the amount of starchy food increased. The amount of water taken at meal-time should be carefully restricted. Beneficial results are often obtained in these cases by the use of *nux vomica* or simple bitters before meals, and the regular administration of hydrochloric acid (gtt. v to viij of the dilute acid) shortly after meals. The general treatment must not be neglected. The patient should lead an out-of-door life as much as possible, and should take regular but very moderate exercise. Great caution is necessary against overfatigue. Iron may be given in most cases during convalescence; but cod-liver oil should be carefully avoided until the gastric symptoms have quite disappeared. Relapses are easily excited, and the most constant care regarding the food must be maintained for months, or even years.

#### DILATATION OF THE STOMACH.

Moderate dilatation of the stomach is quite a frequent condition, although it is not so large a factor in the disorders of digestion in infancy and childhood as many who have written upon the subject would lead us to believe. A very marked degree of dilatation is rare, but in these cases its recognition is important and its treatment difficult.

Dilatation is almost invariably regular or cylindrical; it is usually most marked at the cardiac extremity. Cases of irregular or saccular dilatation, except when associated with cicatricial conditions, are of somewhat doubtful occurrence. The irregular shapes of the stomach found at autopsy dependent upon the contraction of the muscular coats, may be easily mistaken for hour-glass contraction or saccular dilatation.

Dilatation may also result from congenital stenosis of the pylorus. The most important predisposing cause, however, is the muscular atony which accompanies rickets. It is found to a slight degree in almost all

severe cases of rickets. The principal exciting causes are continued distention from overfeeding and chronic indigestion.

In most cases the only symptoms are those of the chronic indigestion which almost invariably accompanies dilatation. The vomiting seen with dilatation is peculiar in that it is infrequent, possibly only once a day; but then the quantity vomited is larger than the last meal taken. In young infants the pressure symptoms resulting from acute dilatation may be very serious. This is particularly true of those with acute bronchitis or broncho-pneumonia, or atelectasis. In such patients I have seen very grave symptoms accompany the rapid distention of a dilated stomach, and in one very delicate infant of three months this was apparently the cause of death. A positive diagnosis of dilatation is only made by the physical signs. There is epigastric fulness and distention, and in some thin patients the outline of the stomach can be distinctly seen. Dilatation of the transverse colon, however, may be mistaken for dilatation of the stomach. In the latter, the lower outline is convex, while in the former it is usually slightly concave. The most satisfactory means of diagnosis is by percussion. The examination should be made three or four hours after feeding, at which time the whole abdomen is apt to be tympanitic. The stomach should then be filled with water; the lower limit of the area of flatness will be the lower border of the stomach. This is much more satisfactory than determining the outline after the generation of gas in the stomach. If the lower border comes below the umbilicus, it is dilated.

In moderate dilatation of the stomach the prognosis is good unless due to pyloric stenosis. If the infant has any acute or chronic pulmonary disease, dilatation of the stomach may add to the discomfort and even to the danger from that condition. The distention of a dilated stomach occurring in the course of any acute pulmonary disease should be relieved by the use of the stomach tube.

In the management of these cases the first point is to restrict the use of fluids, reduce the size of the meals, and regulate the diet in accordance with the general plan outlined in the chapter on Chronic Indigestion. If the dilatation is marked, the stomach should be washed once a day. The general condition of the patient usually requires tonics. Rickets, if present, should receive its appropriate constitutional treatment.

#### ULCER OF THE STOMACH.

Ulceration of the stomach may be found in connection with several pathological processes which are quite distinct from one another:

1. *Ulcers in the Newly Born.*—These have already been referred to in the chapter on Hæmorrhages of the Newly Born. The only characteristic symptom is hæmorrhage.

2. *Ulcers Resulting from Acute Gastritis.*—These also are not frequent. As a rule they give no symptoms except those of gastritis, although in several cases I have known severe hæmorrhage to result from them. This symptom will be considered later.

3. *Tuberculous Ulcers.*—These are quite rare. I met with gastric ulcers five times in one hundred and nineteen autopsies on tuberculous cases; however, the evidence was not conclusive in all of them that the ulcers were tuberculous; but in three the tubercle bacilli were found. Usually there were several small ulcers; in one case but two were present, the larger one being nearly three-fourths of an inch in diameter, and situated on the posterior wall near the middle of the greater curvature. All but one of these cases were in infants, one child being only ten months old. The ulcers gave no symptoms during life, and death took place from general tuberculosis. This is the history of nearly all the few cases on record. In one, however, reported by Casin, a tuberculous ulcer perforated the stomach and caused death from peritonitis.

4. *Simple Perforating Ulcers.*—These are of great rarity and uncertain pathology. I have found but five recorded cases in young children in non-tuberculous patients, two of these being young infants. Rotch's patient was but seven weeks old, and Cade's but two months. Two other cases were under four years old.

The symptoms of ulcer before perforation are gastric pain and tenderness, vomiting of blood, and often bloody stools. In most of these cases in children there were no symptoms until perforation, then followed collapse, sometimes high temperature, the rapid development of tympanites, and death from shock or from peritonitis.

The prognosis is bad in all forms of ulcer of the stomach, except the small follicular variety. In this, however, the diagnosis can not positively be made except by gastric hæmorrhage, and it is only this which makes these cases serious.

**Treatment.**—The treatment is absolute rest, ice by mouth, small doses of opium, and rectal feeding; later, bismuth, arsenic, or nitrate of silver. If symptoms of perforation occur the abdomen should be opened without delay, as offering the only chance of recovery.

## TUMOURS OF THE STOMACH.

Although exceedingly rare, tumours of the stomach occur in childhood, and are seen even in infancy. A case of *sarcoma* of the stomach in a child of three and a half years has been reported by Finlayson. It was apparently primary. The microscopical examination showed it to be of the spindle-celled variety. This writer could find no other recorded case under the age of fifteen.



*Lymphadenoma* of the stomach in a rachitic infant of eighteen months has been recorded by Rolleston and Lathan. There were multiple tumours arising from the mucous membrane in the pyloric region. The case in many features resembled leukæmia.

Six cases of *cancer* of the stomach in children under ten years are collected in an article by Osler and McCrae. Four of these were in young infants and probably congenital. One case, in a child of eight, presented the usual symptoms and lesions of the adult disease.

#### HÆMORRHAGE FROM THE STOMACH (*Hæmatemesis*).

The most frequent variety of hæmorrhage from the stomach, that is seen in the newly born, has already been considered.

I have met with three fatal cases in young infants, the eldest being fifteen months old. In the first case there were symptoms of ordinary gastro-enteritis. On the seventh day the vomiting of blood began, and was repeated about ten or twelve times during the next twenty-four hours, when death took place. The blood was quite abundant, as much as a drachm of red blood being discharged at once. At autopsy there were found in the stomach about two ounces of dark-brown fluid, but no gross lesion was discovered, and no explanation of the bleeding. This hæmorrhage was apparently capillary. In the second case there were symptoms of acute gastro-enteritis of thirty-six hours' duration. After this time there was marked abdominal distention with symptoms of collapse; then a profuse hæmorrhage from the stomach, the child dying while vomiting blood. At least half a pint was discharged. The stomach contained at autopsy two ounces of dark fluid blood, and the mucous membrane was filled with minute ulcers extending quite through the mucosa. In the third case there was no vomiting of blood, but the patient died with symptoms of internal hæmorrhage. There was blood in the upper part of the intestine, and the stomach was filled with blood; it contained many small follicular ulcers resembling those found in the previous case.

Hæmorrhage from the stomach may occur in purpura, hæmophilia, scurvy, and rarely in malaria. In young girls about puberty it may be a form of vicarious menstruation. Occasionally blood may be vomited in cases of hæmorrhagic measles. Two cases are reported in which fatal hæmorrhage followed the swallowing of a foreign body. In both, vomiting of blood occurred long after the original accident. In one case two and a half years had elapsed. The autopsy in this case showed impaction of the foreign body and ulceration into the arch of the aorta. Spurious hæmorrhages may occur where blood has been swallowed and then vomited. The source of this is most frequently the nose or pharynx. It may happen in infants at the breast, where the blood is drawn from

a fissure or ulcer in the nipple. The amount of blood vomited under these circumstances may be large enough to be quite alarming. It may be recognised by the child's general condition being normal, and by the presence of fissures or ulcers upon the nipple. It may sometimes be noticed that the vomiting of blood follows nursing from one breast and not from the other.

**Symptoms.**—There may be no symptoms except those of internal hæmorrhage, but this is rare. Usually there is vomiting of blood, and blood appears in the stools. If the hæmorrhage is rapid and vomiting speedily occurs, the blood may be of a bright-red colour. If it has been long in the stomach it is of a dark-brown or black colour resembling coffee-grounds. The stools containing blood from the stomach are black and tarry in appearance. The general symptoms will depend upon the amount of blood lost.

In a case where blood is vomited, the first point is to distinguish spurious from true gastric hæmorrhage. The nose and pharynx, especially its posterior wall, should be carefully examined. If the child is at the breast, the nipples should be examined. In older children it is important to distinguish vomiting of blood from hæmoptysis. This distinction is to be made in accordance with the rules laid down in text-books on general medicine. The prognosis is bad if the hæmorrhage is due to ulcer, if it is very profuse, or if it occurs in young infants. When it occurs in connection with constitutional diseases the prognosis depends upon the original disease.

**Treatment.**—Altogether the most efficient remedy is the suprarenal extract. It may be given very freely, at least two grains every half hour to a child of one year. The patient should be kept quiet, preferably upon the back; if there are signs of collapse, stimulants may be given hypodermically or by the rectum. No food or water should be given by the stomach for at least twenty-four hours after the hæmorrhage has ceased.

#### THE SWALLOWING OF FOREIGN BODIES.

Between the ages of one and four years the habit of swallowing foreign substances is a very common one. The variety of objects swallowed includes all those articles which the young child can reach and put into his mouth. The most common are detached parts of toys, marbles, pebbles, buttons, and coins. Not only are such smooth articles swallowed, but also with equal readiness, sharp ones, such as pins of every variety, bits of glass, fragments of bone, nails, and small toy knives and forks, etc. At the time of swallowing, choking attacks, severe pharyngeal pain, and sometimes slight hæmorrhage may occur. Symptoms referable to the œsophagus are very few. Nor in the stomach are symptoms often excited. While passing through the intestine there may be colicky pains,

but in the majority of instances there are no symptoms whatever even with sharp or angular bodies. Impaction and perforation, while possible, are extremely rare. The usual time required for a foreign body to traverse the intestinal tract is from four to ten days, but it may be considerably longer. If the body swallowed is a smooth one, it passes the sphincter ani without difficulty. But with sharp bodies there may be severe pain and sometimes hæmorrhage.

The diagnosis is often a matter of much difficulty, and without an X-ray examination a positive diagnosis is impossible. Very often when the physician is called because this condition is suspected by parents the alarm turns out to be a false one.

It is most surprising to see the size, variety, and dangerous character of the foreign bodies which pass through the intestinal tract without causing any symptoms whatever. The expectant treatment is therefore by all means to be recommended. No emetics or cathartics should be administered. The diet should be abundant and composed of articles of food which leave much residue, e. g., coarse cereals, bread, and vegetables. Most of all operation should not be performed or even considered unless there are definite local symptoms.

Quite distinct from such accidental swallowing of foreign substances as has just been described, is the practice of pulling off and swallowing fur from rugs, wool from toys or blankets, shreds from clothing, and a great variety of other substances. This habit is usually seen in nervous children, and often in those where some gastric irritation seems to excite an abnormal craving. In infants the quantity of the substance is generally small and usually it provokes vomiting or the material is speedily passed by the bowel. Very recently in the Babies' Hospital a coloured child of about eighteen months passed in one day a large mass of hair which she had pulled from her own head. Another child in the same ward pulled into shreds and swallowed a large portion of the foot of a cotton stocking, and passed the same by the bowel the following day. Such occurrences are not very common. It occasionally happens that the substance swallowed does not pass the bowel but forms an intestinal tumour which may give rise to obscure and sometimes to severe symptoms of long duration. But more often the tumour forms in the stomach. These gastric tumours are usually composed of hair from the patient's own head. They are more frequently seen in older children than in infants, and usually in girls on account of the long hair. Many of these patients are of the nervous type. The habit may continue until a tumour of considerable size may form, sometimes attaining two or three pounds in weight.

The symptoms are vague until the tumour is discovered. There are usually gastric disturbances of a rather indefinite character. Epigastric pain is common, but vomiting is not especially marked. The general health may suffer but little for a long time. The tumour may be mis-

taken for cancer, a displaced spleen or kidney, faecal impaction, or a tumour of the omentum. A correct diagnosis is seldom made until operation is done. In a few instances the tumour has disappeared after catharsis. If operation is done the outcome is almost always favourable.

## CHAPTER VI.

### DISEASES OF THE INTESTINES.

#### MALFORMATIONS AND MALPOSITIONS.

**MALFORMATIONS** are not very frequent, but are of great variety. With the exception of those situated at the lower end of the intestine they are not of much practical importance, for the condition is such ordinarily as to be incompatible with life. Malformations may be met with at any point in the canal, but most frequently in the rectum and anus. Aside from these, malformations of the large intestine are much less common than those of the small intestine.

**Malformations of the Rectum.**—In Fig. 50 are shown the usual varieties of malformation of the rectum. The most frequent is atresia of the anus (1). In this the cutaneous septum has not been absorbed, but the intestine is normal to its lower extremity. This form is readily curable by a surgical operation. In the next variety (2) the cutaneous orifice and the lower part of the rectum are normal, but a membrane separates this portion from the upper part of the gut; this is usually situated within two or three inches of the anus. The bulging of the lower part of the distended intestine can usually be felt by the finger in the rectum, and a simple division of the membrane by a guarded bistoury may relieve the condition. The third form (3) is more serious. Here the rectum terminates in a blind pouch at a variable distance from the anus, and is represented below by an impervious fibrous cord. The diagnosis of this condition can not positively be made without opening the abdominal cavity. The bulging of the intestine appreciable by the finger in the rectum, is the only point which differentiates the preceding variety from this one. Instead of atresia of the rectum there may be stenosis of varying degrees, which may give rise to the usual symptoms of stricture. This is often curable by dilatation.

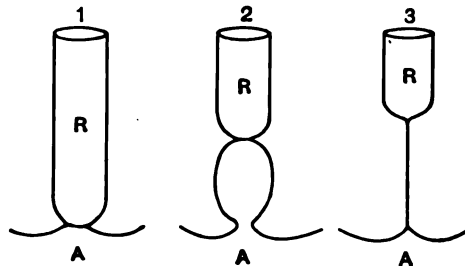


FIG. 50.—MALFORMATIONS OF THE RECTUM.  
A, anus; R, rectum.

**Malformations of the Small Intestine.**—There may be stenosis or atresia at any point, often at many points. Obstruction is much more frequent in the upper than in the lower part of the small intestine, the most common seat being the duodenum. Atresia is more often seen than stenosis. There may be a single point of obstruction, or the lumen of the intestine may be obliterated for a considerable distance, the intestine being represented only by a fibrous cord which connects the two open portions, or there may be no connection between them. In all cases the intestine above is found very greatly distended, while that below is empty and usually atrophied. The causes of these multiple deformities are mainly two—fœtal peritonitis and volvulus. In fœtal peritonitis there are usually found bands of adhesions between the intestinal coils, and between the intestine and the solid viscera. Syphilis has been assigned as a cause in many cases. Volvulus, or a twisting of the intestine during its development, is a more satisfactory explanation for the majority of the cases, especially where there are multiple points of atresia. All these conditions are beyond the reach of surgical treatment. The symptoms appear soon after birth and are those of intestinal obstruction. The higher the point of obstruction the shorter the duration of life; it is rarely more than a week in any case of atresia; in stenosis it may be two or three months.

**Meckel's Diverticulum.**—This is the remains of the omphalo-mesenteric duct, which in fœtal life forms a communication between the intestine and the umbilical vesicle. It is given off from the ileum, usually about a foot above the ileo-cæcal valve. Most frequently it exists as a blind pouch from one-half to two or three inches long, communicating with the intestine. At the extremity of this there may be a fibrous cord, which is free in the abdominal cavity or attached to the umbilicus. In other cases the duct may remain pervious quite to the umbilicus, so that there is a fæcal fistula. Prolapse of the mucous membrane of the duct may lead to an umbilical tumour, described elsewhere. Meckel's diverticulum, especially when present as a cord connecting the ileum with the umbilicus, may compress a coil of intestine, leading to obstruction or even strangulation. This may occur in infancy or later in life.

**Malpositions.**—The ascending colon may be found upon the left side. There may be a complete transposition of the abdominal viscera. In cases of congenital umbilical hernia a large part of the intestines may be found in the tumour, and in diaphragmatic hernia they may be in the thoracic cavity.

#### DIARRHŒA.

The term *diarrhœa* is used to include all conditions attended by frequent loose evacuations of the bowels. These depend upon an increase in peristalsis and in the intestinal secretions.

The importance of diarrhoeal diseases in children can best be appreciated by reference to the following table, showing the mortality of diarrhoeal disease in children under two years, as compared with that from certain infectious diseases for all ages.

*Deaths in New York City for Five Years.*

|  |        |
|--|--------|
| Measles, all ages . . . . .                  | 3,378  |
| Scarlet fever, all ages . . . . .            | 4,152  |
| Pertussis, " " . . . . .                     | 2,000  |
| Typhoid, " " . . . . .                       | 3,523  |
| Diphtheria, " " . . . . .                    | 10,277 |
| Total deaths from five diseases . . . . .    | 23,330 |
| Diarrhoeal disease under two years . . . . . | 26,563 |

There are several important underlying factors upon which diarrhoeal diseases depend. Their greatest frequency belongs to the first year of life; and after the second year a notable diminution both in frequency and severity is seen, and a fatal outcome is relatively rare. The extreme susceptibility in infancy is due to several causes. The digestive organs are severely taxed to provide for the needs of the growing body. The mucous membrane of the gastro-enteric tract of all infants is very delicate in structure, and even in those with good health is exceedingly vulnerable. This vulnerability is enormously increased in the very young, and in those who are feeble, delicate, or suffering from any form of digestive disorder. The mucous membrane of the digestive tract is furthermore constantly exposed to injury, either mechanical or chemical, and to infection.

The next most striking fact about diarrhoeal diseases is their prevalence during the summer season. This is graphically shown in Fig. 51, where are given by months the mortality records for New York City for ten years.

Diarrhoeal diseases are especially frequent in cities and among the poor, for here are found united the three great causes—unfavourable hygienic surroundings, want of proper care, and improper food and feeding. Severe and even fatal cases are, however, met with among all classes and in all places. Their frequency and severity are both increased by want of cleanliness and bad hygiene.

But intelligent care with proper feeding, even in very poor surroundings, may enable children to escape serious diarrhoea in summer.

Everything which lowers the general vitality increases the liability to diarrhoeal diseases. Chronic disorders of digestion, marasmus, malnutrition, and rickets are especially important factors.

Occasionally diarrhoea and dentition are closely associated, the bowels

quickly becoming normal when the teeth have pierced the gum. These cases, although rare, do occasionally occur.

The form of feeding is an etiological factor of the first importance. Of 1,943 fatal cases which I have collected, only three per cent had the breast exclusively. Fatal cases of diarrhoeal disease in nursing infants are rare. It is not, however, artificial feeding *per se* that is to be blamed, but ignorant feeding and improper food. This is shown by the relatively small number of deaths from diarrhoeal diseases seen among the intelligent well-to-do. Breast-feeding requires but little experience, and may be very successfully done even by those with a very low grade of intelligence and among the poor; but artificial feeding is not successful

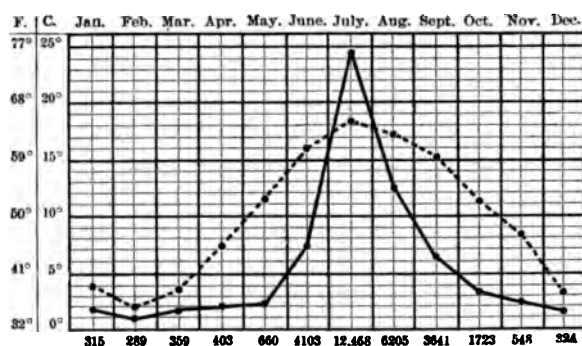


FIG. 51.—MORTALITY FROM DIARRHOEAL DISEASES IN NEW YORK FOR TEN YEARS IN CHILDREN UNDER FIVE; COMPARED WITH THE MEAN TEMPERATURE FOR THE SAME PERIOD. —, mortality; - - - - - , mean temperature. (Seibert.)

unless carried on with much intelligence and experience, and at the same time with a certain amount of money to secure reliable materials, especially pure milk.

That impure milk can cause diarrhoea in infants is a fact that seems established beyond question. I have myself seen every one of twenty-three healthy children, all over two years old, occupying one dormitory cottage, attacked in a single day with diarrhoea, which was traced to this cause. The important question is whether impure milk, especially the bacterial contamination of milk, is the chief cause of the great increase of diarrhoeal diseases in summer, or whether this is only one of the factors, others, especially atmospheric heat, being really more important. Since about the year 1890, when the enormous bacterial contamination of milk began to be appreciated, the opinion has prevailed that in this was to be found the real cause of the prevalence and fatality of diarrhoeal diseases in summer. This belief carried with it the expectation that by furnishing to every artificially fed infant a clean, fresh milk, or milk which had been pasteurised or sterilised, this great cause of infant mortality could largely be removed. It is true that a

great reduction in infant mortality from summer diarrhœal diseases has been effected during the last two decades; but it is also true that there has been quite as great a reduction in infant mortality in other seasons, and, in summer, from other causes than diarrhœal diseases. (See Fig. 52.) This leads us to raise the question, whether the assumption that

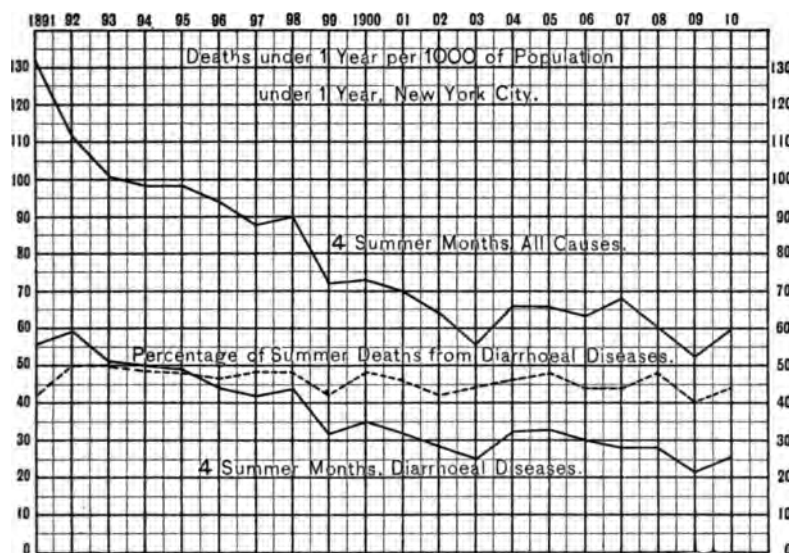


FIG. 52.—DEATHS UNDER ONE YEAR PER 1,000 OF POPULATION UNDER ONE YEAR, NEW YORK CITY. A comparison of summer deaths from all causes with summer deaths from diarrhœal diseases for a period of twenty years.

the bacterial contamination of milk is the great cause is correct, and also whether the lowered mortality in summer has not been brought about quite as much by other conditions, such as better hygiene and care and a better understanding of infant-feeding, as by the exclusion of germs from milk or their destruction by heat.

In the years 1901 to 1903 an investigation<sup>1</sup> was undertaken by The Rockefeller Institute in co-operation with the Health Department of New York to secure data regarding the following points: (1) The results in infant-feeding obtained with milk of different degrees of purity both in winter and in summer, as shown by the gain or loss in weight, the amount of gastro-intestinal disturbance, and the death rate; (2) the relation, if any, existing between the number of bacteria present in the milk and the frequency of diarrhœal disease; (3) whether any organisms with pathogenic properties could be found in milk to which diarrhœal disease could be ascribed as a cause; (4) whether the practice of heating milk—pasteurisation or sterilisation—affected the results ob-

<sup>1</sup> The full report of this investigation was published by Prof. William H. Park and the author in the Medical News, December 5, 1903.



tained with any given milk; (5) to what degree older children as well as infants were affected by bacterial contamination of milk.

Observations were made upon 592 bottle-fed infants living in tenements of New York; 202 were observed in winter and 390 in summer. The infants were well when the observations were begun, and were watched for a period of about three months, being visited regularly by physicians, who gave advice when needed. For some of the children no change was made in the milk which they were already taking; for others special milk was provided. Samples of milk as fed to the children were frequently examined as to the number and character of the bacteria present. Observations were possible upon infants taking (1) condensed milk, (2) the cheapest grade of store milk, such as is usually purchased by the poor, (3) a better grade of milk delivered in bottles, (4) the best bottled milk sold in the city, all of the above being prepared at home, (5) milk modified at milk depots and furnished to patients in separate feeding-bottles.

During the winter period of observation, the mortality was but 2.5 per cent, and in but one instance was death due to disease of the digestive tract. The health of the infants observed was not appreciably affected by the kind of milk nor by the number of bacteria which it contained. The different grades of milk varied much less in the amount of bacterial contamination in winter than in summer, the cheap store milk averaging only about 750,000 per c.c.

During the summer period, the mortality was 10.5 per cent, four-fifths of the deaths being due to diarrhoeal disease. The worst results were seen in those whose food was either the cheap grade of store milk or condensed milk, and in those who received the poorest care. The best results were seen in those whose food was the best grade of bottled milk, or modified milk furnished from milk depots, and in those who received the best care.

The number of bacteria which milk may contain before it becomes noticeably harmful to the average infant in summer is not at all uniform. Of the usual varieties present, no strikingly deleterious results were seen until the number approached the one million mark. But much above this point injurious effects were usually manifest. Below it other factors rather than the number of bacteria seemed of greater importance in producing diarrhoea. Thus in condensed milk, prepared as it usually was with hot water, the bacterial contamination was relatively small, yet the results were almost as bad as with the most highly contaminated milk.

No relationship could be discovered between any special forms of bacteria present in the milk and the health of children. The pathogenic properties of 139 varieties of bacteria isolated from milk were tested upon animals in various ways, chiefly by feeding pure cultures to young

kittens. The results were negative. Nor could a relationship be established in any other way between any special form of bacteria in milk and the summer diarrhœas of infancy.

To test the effect of heating milk, observations were made during two summers upon 92 infants who were taking the modified milk prepared at a milk depot. The milk used was from a good farm, and had been kept properly cooled. The infants were divided into two groups as nearly alike as possible. To one group the milk was given pasteurised (165° F. for thirty minutes), to the other group the same kind of milk was given raw. All the infants were well at the beginning of the period of observation. The results are shown in the following table:

| Food.   | Total number of infants. | Remained well entire summer. | Had severe diarrhœa. | Average days diarrhœa. | Deaths. |
|---|--------------------------|------------------------------|----------------------|------------------------|---------|
| Pasteurised milk containing 1,000 to 50,000 bacteria per c.c. at the time of use..... | 41                       | 31                           | 10                   | 4                      | 1       |
| Raw milk containing 1,200,000 to 20,000,000 bacteria per c.c. at the time of use..... | 51                       | 17                           | 34                   | 11½                    | 2       |

Thirteen of the fifty-one infants on raw milk were changed before the end of the season to pasteurised milk because of serious diarrhœa; but for this the results with raw milk would have been even more unfavourable. A similar experiment was made a third season with almost identical results. Although the number of cases is not large, the results, which were practically uniform for three successive seasons, show unmistakably that in hot weather raw milk, although from a good source, but at the time of feeding highly contaminated with bacteria, causes illness in a much larger number of cases than when it has been previously heated.

After the first two years, children are much less affected by bacteria in milk. The observations seemed to show that milk from healthy cows, produced under cleanly conditions and kept at a temperature below 60° F., although containing large numbers of bacteria, sometimes amounting to many millions per c.c., might be taken in considerable quantities and for long periods by children over three years old, without any appreciably harmful effects resulting either from the living bacteria or their toxins. A single example is typical of a number of observations made. An orphan asylum, containing 650 children from three to fourteen years old, used during an entire summer milk in which the bacteria ranged from 2,000,000 to 20,000,000 per c.c.; yet during this period there occurred no case of diarrhœa of sufficient severity to call a physician. The milk was kept cold (below 60° F.) until used; but was given without sterilisation.

Mere numbers of bacteria certainly appear to count for much less than was once supposed. But the fact should not be overlooked that milk abounding in bacteria because of careless handling is also always liable to contain pathogenic organisms derived from human or animal sources. These observations, continued for three seasons and giving each summer nearly identical results, indicate that we are to seek elsewhere than in a moderate bacterial contamination of milk the great cause of summer diarrhœas. Though it is clear that excessive contamination is highly detrimental to infants, we must certainly look to the other factors for the explanation of a very large, possibly the largest, proportion of the cases. Of the other factors, atmospheric heat is clearly first in importance. This may act by so interfering with normal digestion and metabolism as to lead to the formation within the body of injurious substances which excite diarrhœa; or it may favour the excessive growth of bacteria ordinarily present in the digestive tract. In this group of cases the rôle of the bacteria seems to be secondary, though perhaps a very important one. According to this hypothesis, the cause of the diarrhœas under consideration is not something introduced from without, but something produced within the body itself.

Another group of diarrhœal diseases exists which may be due to infection introduced from without, through water, milk, or other food; to these the term dysentery is more often applied. These cases have been found to be associated with definite bacteria or amœbæ. It is likely that intestinal disease of this type may supervene upon the preceding one.

#### ACUTE INTESTINAL INDIGESTION AND INTOXICATION.

The cases included in this chapter comprise many types which, however, are closely allied and shade into one another. Though the extremes of the series differ as widely as possible, yet intermediate types of almost every grade are met with. They are discussed under a single heading, since they have no essential anatomical differences, nor, so far as yet determined, do they differ etiologically. Some of the attacks are so mild in character that in children with normal resistance, and receiving prompt treatment, they may last but a few hours. On the other hand, they may be so rapid in development and so severe as to result in death in a few hours; or, beginning with less intensity, they may be the starting point of prolonged functional disorders or may prepare the way for the development of infectious processes.

**Etiology.**—The most important causes have been mentioned in the foregoing discussion on the General Etiology of Diarrhœal Diseases. A predisposition to attacks is furnished by summer weather, a delicate constitution, and any previous derangement of digestion. The exciting cause of an attack may be the use of improper food, overfeeding or some sudden

change in food as in weaning; but, the food remaining unchanged, it is often other influences affecting the child, such as summer heat. The most striking thing about these cases is their prevalence during hot weather. Year after year are repeated in New York the conditions which are graphically represented in Fig. 51, viz., an epidemic which, beginning in June, rapidly increases in severity, reaching its height usually in July, from which time it diminishes steadily, regularly coming to an end in September. What is true of New York is true also of Philadelphia, Chicago, and other large cities of the temperate zone. The severity of the epidemic bears a fairly close relation to the height of the summer temperature. Thus in Chicago and Philadelphia, of the deaths under one year, 32 per cent are due to acute gastro-intestinal diseases; in New York, 27 per cent; in Boston, 19 per cent; in London, only 13 per cent. A comparison of the mortality and temperature curves shows that while the mean temperature rises gradually during April and May, it is not until a certain temperature is reached, that any notable increase in diarrhoeal diseases begins.

Despite the fact that since 1886 many series of bacteriological studies of the intestinal discharges have been made by Booker and Park in this country, by Baginsky, Escherich, and others in Germany, our knowledge of this subject is still very incomplete. The conditions are exceedingly complicated, and the problem is a very difficult one. So far as is now known, no one form of bacteria can be assigned as the cause of this group of diarrhoeas. There seems to be evidence that the Shiga bacillus may produce diarrhoeal disease which clinically does not differ from this type. But it is wanting in so large a proportion of cases, that it can not be regarded as a specific cause. With existing knowledge it seems probable that there are a number of organisms present in the intestine in disorders of digestion, which, under favourable conditions, may multiply to such a degree as to produce serious disturbances. But the rôle of the micro-organisms may be regarded as a secondary one.

There are certain cases in which toxic symptoms of a severe type develop abruptly in children previously quite well. These only are to be regarded as examples of acute milk poisoning. Although the bacteria in the milk may have been previously destroyed by sterilisation, the toxins produced by them may still be present. This is doubtless the explanation of the simultaneous development of several cases in families or institutions.

We can not believe that direct contagion is the usual way in which this disease is spread. When occurring in institutions or in families, it usually happens that a number of children are attacked simultaneously rather than successively, this indicating a common cause, usually to be found in the food, the surroundings, or the atmospheric conditions.

**Lesions.**—In the milder cases which end in recovery, the anatomical changes are negligible. In those which prove fatal from the disease itself, or from some associated condition, the lesions are, in brief, a superficial catarrhal inflammation affecting the entire gastro-enteric tract, but varying much in severity in the different regions and in the different cases. The colon, the lower ileum, and the stomach are apt to suffer most, the duodenum and the jejunum least.

*The Gross Appearances.*—These may show but little that is abnormal. The walls of the stomach may be coated with mucus, and the mucous membrane may show intense congestion, generally in patches, or it may be pale. The mucous membrane of the small intestine may be pale throughout; there are often irregular areas of congestion, or a very intense congestion of a large part of its surface, particularly in the ileum. With this there may be redness and swelling of Peyer's patches and the solitary follicles. In the colon the mucous membrane is congested, especially upon the rugæ. The solitary follicles are usually swollen. The changes described are not at all uniform, and do not differ very greatly from the appearances often seen in the intestines when patients have died of other diseases.

In the cases classed clinically as cholera infantum, the pathological changes are more characteristic. The greater part of the small intestine, and sometimes the entire colon, are distended with gas, and contain material of a grayish-white colour about the consistency of a thin gruel. It has a mawkish odour, but usually not a very offensive one. The mucous membrane of the entire intestinal tract has in most cases a pale, "washed-out" appearance. Sometimes this is seen only in the small intestine, while there are areas of congestion in the colon. If cholera infantum has been engrafted upon some other pathological process in the intestines, as is not infrequent, there is found post-mortem evidence of this in the form of severe catarrhal inflammation, sometimes old ulcerations.

*The Microscopical Appearances.*—Unless autopsies are made very soon after death—at most within four hours—it is not safe to draw conclusions from the conditions found, as post-mortem changes take place readily, and resemble those of the disease under consideration. This applies particularly to the condition of the epithelium.

The essential lesion consists in degenerative changes in the epithelium of the stomach and intestines. The cells may still be present, but with the cell protoplasm and nuclei so changed that they do not stain normally. In more severe and prolonged cases the superficial epithelium in places is entirely destroyed; these changes mark the beginning of ileo-colitis.

The changes in and about the blood-vessels are variable. The small vessels may be distended, and there may be hæmorrhages or an exuda-

tion of leucocytes in their neighbourhood. These appearances are seen either in the mucous or submucous layer. Peyer's patches and the lymph nodules may be enlarged from cell-proliferation. Pathologically no sharp line can be drawn between these lesions and those of the early stage of ileo-colitis; the latter affect the lower ileum and colon chiefly, often exclusively, are more advanced, and involve the deeper parts of the intestinal wall.

*Lesions in Other Organs.*—These are much less frequent and less severe than in the more protracted cases of ileo-colitis. Acute bronchitis and broncho-pneumonia are frequent. Acute degeneration of the kidney is found to some degree in every case which is severe enough to cause death, and in a few there is acute nephritis. In rare cases a general septicæmia, due most frequently to the streptococcus, is present. Degenerative changes are sometimes found in the liver cells, and even in the nervous centres.

*Symptoms.*—Clinically, these cases may be divided into three groups: (1) The mild form, with definite local symptoms, but few general ones; they may be of short duration or protracted; (2) the severe form in which there are not only local but marked constitutional symptoms, fever, etc.; (3) cholera infantum, the more severe and fatal type met with.

*The Mild Form.*—In infants, acute indigestion is seldom limited either to the stomach or to the intestine, although in one case the disturbance of the stomach is slight and that of the intestine serious, and in another the reverse may be observed. In these little patients the intestinal symptoms are more frequent, and, as a rule, more severe than those referable to the stomach. In older children it is not uncommon to see the intestinal symptoms alone. In infants, if the attack develops suddenly, gastric symptoms are usually present; if more gradually, they are usually absent. The local symptoms are colicky pain, tympanites, and later diarrhœa. The constitutional symptoms, prostration and nervous disturbances, are slight or absent. Pain is indicated by the sharp, piercing cry, great restlessness, and drawing up of the legs. Tympanites is rarely very marked. The stools are always increased in number and are from four to twelve a day. If more frequent they are very small. The first stools are more or less fecal, but this character is soon lost. The colour is at first yellow, then yellowish-green, and finally often grass-green. This colour is due to biliverdin. If the child has been taking milk, masses of undigested milk, chiefly fat, are present. The reaction of the stools is almost invariably acid. The odour may be sour, or it may be foul. The stools are much thinner than normal, and often frothy from the presence of gases. Blood is not present, nor is much mucus seen, unless the symptoms have lasted several days. The microscope shows, in addition to food-remains, epithelial cells, usually of the

cylindrical variety, which are numerous in proportion to the severity and duration of the attack. The bacteria are the ordinary forms found in the faeces.

The course and termination of the disease depend upon the previous condition of the patient, the nature of the exciting cause, and the treatment employed. In a previously healthy child, if the cause is at once removed and proper treatment instituted, the severe symptoms rarely last more than a day or two, and in four or five days the patient may be quite well. In delicate infants, a severe attack of acute intestinal indigestion in the hot season is likely to prove the first stage of a pathological process which may continue until serious organic changes in the intestine have taken place. This result may not follow the first attack, but one is often succeeded by others until it occurs. If circumstances are such that proper dietetic treatment and general hygienic measures can not be carried out, this termination is very common.

In older children most of the cases seen are of the milder type. The onset is often with vomiting; pain is generally mild and precedes diarrhoea by several hours. It is seldom localised but is more often referred to the navel. The stools are loose, frequent, and contain undigested food, and are of almost every conceivable colour and variety. The temperature, if elevated at all, is so only for a short time. There is general anorexia and a coated tongue. With proper treatment the attack is usually over in a few days, being very seldom followed by the severer types of diarrhoea, as is so commonly the case with infants.

*The Severe Form.*—This may follow after several days of an apparently mild attack, especially during hot weather or if improperly treated. In the cases developing suddenly, the clinical picture is quite a definite one.

An infant is restless, cries much, sleeps but a few minutes at a time, and seems in distress. The skin is hot and dry, the temperature rises rapidly to 102° or 103° F., sometimes to 106° F., and all the symptoms indicate the onset of some serious illness. He may lie in a dull stupor, with eyes sunken, weak pulse, and general relaxation, or there may be restlessness, excitement, and even convulsions. There may be great thirst, so that everything offered is eagerly taken, or everything may be refused. Vomiting may be an early and important symptom. It is first of food, often that which was taken many hours before; retching continues even after the stomach has been emptied, so that mucus, serum, and sometimes bile may be ejected. Vomiting does not usually persist throughout the attack, and in many cases it is absent altogether. Diarrhoea is sometimes delayed for twenty-four hours or even longer after the beginning of the grave constitutional symptoms. At first there are faecal stools, then great bursts of flatus, with the expulsion of a thin

yellow material with an offensive odour. Four or five such discharges may occur in as many hours. At other times the stools are gray, green, or greenish-yellow, and sometimes brown. The characteristic features are the amount of gas expelled, the colicky pains preceding the discharges, and the foul odour. After the first day the stools may be almost entirely fluid, varying in number from six to twenty a day, and often large even then. Their offensive character usually continues. After two or three days mucus appears. The microscopical examination of the stools shows great numbers of separate epithelial cells, and sometimes groups of cells attached to a basement membrane. In addition there may be leucocytes and some red blood-corpuscles.

In many cases the free evacuation of the bowels is followed by a drop in the temperature and subsidence of the nervous symptoms, and the child may fall asleep. The prostration, though often great in the beginning, may not be of long duration. Under the most favourable circumstances, after one or two days of severe symptoms, convalescence may take place. The stools continue frequent for five or six days, but gradually assume their normal character, and recovery follows. The chief factors contributing to such favourable results are a good constitution on the part of the child, prompt and intelligent treatment at the outset, and proper feeding afterward.

If the circumstances are not so favourable, if the patient is a very young or delicate infant, there may be no reaction from the first severe symptoms, and the attack may terminate fatally in from one to three days. In such cases the temperature remains high; the stomach may or may not be disturbed; but the diarrhoea, prostration, and nervous symptoms continue, and death occurs from exhaustion, in coma or convulsions. Instead of a rapidly fatal termination, the severity of the early acute symptoms may abate somewhat, and the attack assume the character of ileo-colitis, with a lower but continuous temperature of 100° to 102° F., frequent mucous stools, wasting, etc. The urine is scanty and concentrated, and in most of the severe cases with very high temperature contains a small amount of albumin, and occasionally a few hyaline and granular casts. These are the result of degenerative changes in the renal epithelium. In rare cases there are evidences of acute nephritis. Broncho-pneumonia is sometimes seen.

It not infrequently happens, after the storm of the acute attack with its high temperature, intense prostration, and grave nervous symptoms is passed, and the stools are so much improved that the patient is regarded as out of danger, that all the former symptoms may develop with such rapidity and severity as sometimes to carry off the patient in from twelve to twenty-four hours. Such relapses are generally excited by some mistake in the diet, usually that of allowing milk too soon. The amount of milk given may be small, and yet the symptoms follow its



administration so soon that there can be little doubt regarding the connection between them (Fig. 53). Besides such severe cases, many

milder relapses are seen; the cause is usually some error in diet.

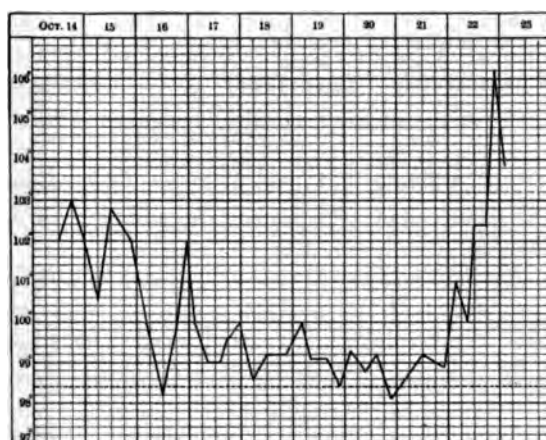


FIG. 53.—ACUTE INTESTINAL INTOXICATION WITH FATAL RELAPSE. Infant five months old; early symptoms, both intestinal and nervous, severe; rapid improvement followed stopping milk, free catharsis and irrigation. After stools had been nearly normal for three days relapse occurred, apparently from adding milk to the diet, although less than two ounces a day were given. Autopsy: Only mild intestinal lesions were present; other organs essentially normal.

*Cases without Diarrhœa.*—Attacks of acute intestinal indigestion with severe intoxication in which there is no diarrhœa, but constipation instead, are most puzzling and frequently most serious. Fortunately, they are not of common occurrence. I have, however, seen several striking examples with very high temperature, grave nervous symptoms, and sometimes marked

abdominal distention in which it seemed almost impossible to move the bowels by drugs. Castor oil, calomel, and salines have in some cases been tried in succession in four or five times the ordinary doses without the slightest effect, even when supplemented by frequent intestinal irrigation. It has sometimes been nearly two days before free movements were finally produced. These are often exceedingly foul. It is somewhat difficult to explain such cases. There seems to exist for the time almost complete intestinal paralysis. The toxic materials are locked up in the small intestine, for the colon is frequently quite empty. When one meets such a case he can appreciate the fact that diarrhœa is a conservative process of the greatest possible value.

In children over two years old there are seen some features which differ from those of the cases above described as occurring in infants. The attacks are more often due to other causes than to milk. Vomiting does not occur so readily as in infants, pain is a more prominent symptom, and the temperature, as a rule, is lower. The nervous symptoms are much less prominent. Skin eruptions, however, are more frequently seen, particularly urticaria, which is a feature of most severe attacks, and in obscure cases has some diagnostic value. Although often beginning with severe symptoms, these cases usually make good recoveries;

there is much less danger of their going on to the development of ileocolitis than in the case of infants.

**Diagnosis.**—The acute indigestion manifested by vomiting and diarrhoeal stools which marks the beginning of so many febrile diseases in infancy, particularly scarlet fever, pneumonia, malaria, and influenza, is often difficult to distinguish from more severe attacks with intestinal intoxication. The question to decide is whether the digestive symptoms are the cause or the result of the fever. It is sometimes not until the case has been watched for some time that one can be certain. Usually where digestive symptoms are secondary they diminish after the first day or two, although the severity of the general symptoms may steadily increase. Where the nervous symptoms are prominent at the outset, it is sometimes difficult to exclude meningitis. I have seen many cases where great doubt existed for several days. One should always hesitate to make a diagnosis of meningitis when marked diarrhoea is present.

**Prognosis.**—Attacks of intestinal indigestion do not often prove fatal, except in young infants or those already suffering from malnutrition. In all cases the prognosis depends upon the previous health of the child, his surroundings, the season of the year, and whether or not the case receives prompt and proper treatment. A continuously high temperature and severe nervous symptoms are bad prognostic signs. The existence of rickets, pertussis, or any other disease, greatly increases the gravity of the attack.

**Prophylaxis.**—A better understanding of the etiology brings with it great possibilities in the prevention of this disease.

Prophylaxis must have regard, first, to the hygienic surroundings of children, and to all sanitary conditions of cities. City children should be sent to the country, whenever it is possible, for the months of July and August. Where a long stay is impossible, day excursions do much good. The fresh-air funds and seaside homes have done much in New York to diminish the mortality from diarrhoeal diseases.

The second part of prophylaxis relates to food and feeding. Maternal nursing should be encouraged by every possible means. Nothing is better established than the close relation existing between artificial feeding and diarrhoeal diseases. Yet, as stated elsewhere, it is not artificial feeding *per se*, but ignorant and improper feeding. Among infants in private practice who are properly fed these attacks are not common.

Overfeeding is particularly to be avoided during days of excessive heat. It is at such times an excellent rule with infants to diminish each feeding by at least one-half, making up the deficiency with water, and to give water very freely between the feedings. In summer all water given to infants or young children should be boiled. Children, like adults, require less food in very hot weather, but more water. Infants cry more from thirst and heat than from hunger, and even those at the breast are

likely to be given too much food. Infants should never be fed more frequently, but always less frequently, during hot weather.

A very important work in practical philanthropy among the poor of our large cities in summer is to provide means for supplying pure milk to infants. This has been done on a large scale in many American cities, and it is one of the important agencies that have affected a decided reduction in the death-rate from diarrhœal disease. It is not enough to furnish to the poor a pure, clean milk in bulk, or even in sealed quart bottles. The advantages of such milk may be entirely lost by the way in which it is cared for in the home or the way in which it is fed to infants. Since the milk must usually be kept at home without ice, sterilisation at 212° F. is advisable. When milk is distributed from milk depots, a physician should be in charge who can keep a general supervision over the children, and advise as to the quantity of food, number of feedings, and the formula to be used. His work should be supplemented by visits of nurses to the homes of patients. An essential feature is to keep such close supervision over the infants as to recognise at once and promptly treat slight disturbances of digestion.

But even more important than pure milk is the education of the poor in all matters relating to infant feeding and hygiene. In no way can this educational work better be done than in connection with milk distribution.

**Hygienic Treatment.**—If the attack is a severe one and occurs in the excessive heat of midsummer, and does not readily yield to treatment, the child should, if possible, be sent to a cooler place. Convalescent cases should also be sent away on account of the dangers of relapse. Usually the seashore is to be preferred to the mountains, but this is not so important as that the child shall go where it can be taken most quickly and can secure the best food and the best surroundings. Children must not only be sent away; they must be kept away until quite recovered. In cases which have become somewhat chronic, more can sometimes be accomplished by a change of air than by all other means.

Fresh air is of the utmost importance for all diarrhœal cases in summer. No matter how much fever or prostration there may be, these children do better if kept out of doors the greater part of the day. Nothing is so depressing as close, stifling apartments. Children should be kept quiet, and especially should not be allowed to walk, even if they are old enough and strong enough to do so. They can be kept out in carriages, in perambulators, or in hammocks.

The clothing should be very light flannel; a single loose garment is preferable. Linen or cotton may be put next the skin if this is very sensitive and there is much perspiration. At the seashore and in the mountains, care should be taken that sufficient clothing at night is supplied.

Bathing is useful to allay restlessness, as well as for the reduction of temperature. For the latter, only the tub bath can be relied on. The temperature of the bath should be about 100° F. when the child is put into it, and should then be gradually reduced to 80° or 85° F. by adding ice. The bath should be continued, with gentle friction of the body, for from five to twenty minutes.

Scrupulous cleanliness should be secured in the child's person and clothing. Napkins, as soon as soiled, should be removed from the child and from the room and placed in a disinfectant solution. Excoriations of the buttocks and genitals are to be prevented by absolute cleanliness and the free use of some absorbent powder, such as starch and boric acid.

**Dietetic Treatment.**—It is of the first importance to remember that during the early stage of the acute cases, digestion is practically arrested. To give food at this time, manifestly can do only harm.

In nursing infants the severe forms of the disease are extremely rare; but the breast should be withheld so long as a disposition to vomit continues, and no food whatever given for at least twenty-four hours. Thirst may be allayed by giving frequently, but in small quantities, boiled water or thin barley or rice water. If these are refused or vomited, absolute rest to the stomach will do more than anything else to hasten recovery. After the stomach has been allowed to rest for twenty-four hours, it is generally safe to permit a nursing child to take the breast tentatively. The intervals of nursing should not be shorter than four hours, and the amount allowed at one feeding should not be more than one-fourth the usual quantity. This may be regulated by allowing an infant to nurse at first only two or three minutes. Between the nursings may be given boiled water or barley water. Nursing may be gradually increased, so that in three or four days the breast may be taken exclusively. If there is any reason to suspect the quality of the breast-milk, such as menstruation or pregnancy, it may be necessary to stop the nursing for a longer time.

In infants under four months who are being artificially fed, all food, and especially milk, should be stopped at once. Milk should not only be withheld during the period of acute symptoms, but for several days thereafter. Besides the articles mentioned above as suitable for the period of most acute symptoms the following substitutes for milk will be found useful: rice or barley water or whey; the farinaceous foods, and broth or bouillon made of veal, chicken, mutton, or beef. Water may be allowed freely at all times unless there is much vomiting.

When milk is begun it should be remembered that the fat is more likely to disturb digestion than any other element. For this reason skimmed milk, fat-free milk, buttermilk, or condensed milk, are useful. The first three mentioned should be sterilised. At first they should be well diluted and very gradually increased in strength. (For

details, see article on Feeding.) Wet-nurses are not to be employed during the acute symptoms, but during the period of prolonged malnutrition which follows an acute attack they may be of the greatest service.

The same general principles of feeding should be applied in older children. All food is to be withheld until the vomiting ceases, when broths and beef juice may be given; later, buttermilk or kumyss and sterilised skimmed milk, or thin gruels. Solid food should not be allowed for several days after the stools have become normal.

**Medicinal and Mechanical Treatment.**—It must be borne in mind that we are not treating an inflammation of the stomach or intestines, although such may be the ultimate result of the process. The essential condition, it should be remembered, is one of indigestion and intoxication arising from the intestinal contents—food-remains from arrested digestion, altered secretions, acids, irritating and toxic substances produced by chemical and bacterial action—to which not only the constitutional but the local symptoms are chiefly due. We can hardly do better than to imitate and assist Nature in her treatment of this condition. Let us consider what this is. Lest too much food be swallowed, appetite is taken away; by vomiting, the stomach is emptied; to neutralise the acid poisons in the intestine, an alkaline serum is poured out from the intestinal walls; to remove irritant poisons, increased peristalsis is excited.

The first indication is, therefore, to evacuate the stomach and the entire intestinal tract at the earliest moment, and to do this as thoroughly as possible. Under no circumstances should the treatment be begun with the use of measures to stop the discharges. To empty the stomach is not necessary in every case, since the initial vomiting may have done this effectively. Whenever vomiting persists one should immediately resort to stomach-washing. A single washing is generally sufficient, and if employed at the outset may do much to shorten the attack. With high fever and great thirst, it is often advisable to leave a few ounces of water in the stomach. If the vomited matters have been very sour, ten grains of bicarbonate of soda may be introduced with the portion which is to be left behind. As a substitute for stomach-washing in children over two years old, or where it can not be employed, copious draughts of boiled water may be given. This is taken readily, and as it is usually vomited almost at once it may cleanse the stomach thoroughly; but it is inferior to stomach-washing.

To clear out the small intestine, only cathartics are available. For the colon, we may in addition employ irrigation. Calomel, castor oil, or the salines may be used as cathartics, and enough of any one of them must be given not simply to move the bowels, but to clear out the intestinal tract thoroughly. There is little danger from too free purgation at the outset. Calomel has the advantage of ease of administration:

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one-fourth of a grain should be given every fifteen or twenty minutes up to six or eight doses. When the stomach is not disturbed, I prefer castor oil in most cases, as it sweeps the whole canal, causes little griping, is very certain, and its after-effects are soothing. Two drachms should be given to a child six months old, and half an ounce to one of four years. Of the salines, the best are the sulphate of soda and Rochelle salts; from one to three drachms may be given, well diluted, divided into four or five doses, at twenty-minute intervals.

The occasional use of cathartics is an important part of the later treatment. Whenever there are signs of an accumulation, or fresh symptoms of intoxication develop, such as increase in temperature, nervous symptoms, etc., another thorough cleaning out of the intestinal tract is indicated. The accumulation may not be the result of food, but simply of intestinal secretions. So long as the processes of fermentation and decomposition continue active, the indications are to facilitate elimination, not to check the discharges.

Early irrigation of the colon is advisable in all cases, as it hastens the effect of the cathartic and removes at once much irritating and offensive material. It should be done two or three times the first day, but afterward once daily is generally sufficient. A saline solution (one tablespoonful of salt to two quarts of water), at a temperature of about 100° F., is to be preferred; and a rectal tube well inserted should always be used. Thorough initial evacuation, no food, but plenty of water for twenty-four hours, and careful feeding after that time, are all the treatment that is necessary in most cases.

Other drugs are of secondary importance. Their value is certainly very much overestimated. It may be questioned whether as yet any proper antiseptic treatment of the gastro-enteric tract is possible.

Of the drugs which are used to influence the intestinal process, bismuth is to be preferred. It has the advantage that it rarely causes vomiting, and that most of its preparations can be given in large doses. The subcarbonate is the safest. It may be given in doses of from five to fifteen grains every two hours, to a child of one year. Like the subnitrate it is insoluble and is best given suspended in mucilage. It usually blackens the stools. It may be kept up throughout the attack. The best results seen from acids are in the later stages and in the sub-acute cases; of the dilute hydrochloric acid, from four to ten drops may be given, best alone, but well diluted. Alkalies are of value only in the acute stage, especially where there is acid fermentation in the stomach, with vomiting and eructations of gas. Lime-water, bicarbonate of soda, magnesia, or chalk-mixture may be employed. My own experience leads me to place little reliance upon astringents. They do little good, and often much harm. They are indicated only in the catarrhal diarrhœa which often follows the symptoms of acute intoxication, but may be

advantageously used in this condition in combination with opium. A useful astringent is tannalbin, which may be given in two-grain doses every two hours to an infant of one year.

While opium in some form is required in many cases, as often used it undoubtedly does great harm. The chief indications for opium are great frequency of movements and severe pain. It is contraindicated until the intestinal tract has been thoroughly emptied by cathartics and irrigation; also when the number of discharges is small, particularly if they are very offensive; it is especially to be avoided in the early stage of very acute cases, and never to be given when cerebral symptoms and high temperature coexist with scanty discharges. Opium is admissible in the early part of the disease after the tract has been thoroughly emptied. It is particularly indicated when there is a persistence of large, fluid movements attended by symptoms of collapse, and in all cases approaching the cholera-infantum type. In such circumstances morphine should be given hypodermically, one-sixtieth of a grain to an infant of six months, to be repeated in two hours if no effect is seen. Opium is useful during convalescence, when the administration of food is immediately followed by a movement of the bowels; and when without an elevation of temperature, often with good appetite, the stools are frequent and contain undigested food, because peristalsis is so active that the intestinal contents are hurried along with such rapidity that there is not time for complete intestinal digestion and absorption. Nothing requires nicer discrimination than the use of opium in diarrhoea. It is wise to administer it always in a separate prescription, and never in composite diarrhoeal mixtures. The dose should be regulated according to its effect upon the number of stools. Enough is to be given to produce a distinct effect—the diminution of pain and the control of excessive peristalsis—but never enough to check the discharges entirely, or to cause stupor. The uncertainty of absorption must also be remembered; a second full dose should not be given until a sufficient time has elapsed for the effect of the first to pass away. For an average child of one year, five minims of paregoric, one-fourth minim of the deodorised tincture, or one-fourth grain of Dover's powder, may be used as an initial dose, to be repeated every one, two, or four hours, according to the effect produced.

Stimulants are often required in severe cases. The prostration is great and develops rapidly; frequently almost no food can be assimilated for twenty-four or thirty-six hours, while the drain from the discharges continues. The general condition of the patient is the best guide as to the time for stimulation and the amount required. Old brandy is the best preparation for general use. An infant a year old may, as a maximum, take half an ounce of brandy in twenty-four hours. Stimulants should always be diluted with at least eight parts of water, and be given in small quantities, at short intervals.

In cases of extreme prostration, the hot bath, mustard to the extremities, and sometimes the mustard pack, are beneficial. When the drain is rapid and very great, and in all cases approaching the cholera-infantum type, subcutaneous saline injections should be used, in the manner described under Cholera Infantum.

**Finkelstein's "Food Intoxication."**—In the chapter upon Difficult Feeding we have already referred to this author's classification of cases indicating different degrees of nutritional disturbance. The most severe form, which by him has been given the name of *food intoxication*, can more properly be discussed in the present chapter. Finkelstein has shown that the causative factor in these cases is not bacterial infection but a failure in metabolism, and that the condition is aggravated and continued by the ingestion of fat and sugar. The various symptoms seen in this condition have for some time been well known, but the credit belongs to Finkelstein of demonstrating their association in a single clinical type. These symptoms do not arise in healthy infants, but in those who have previously suffered from minor disturbances of digestion and nutrition, usually for some time. Occasionally they may develop in the course of some one of the general infectious diseases.

In a marked case with fully developed symptoms the characteristic clinical manifestations of this condition are: (1) certain nervous symptoms, sometimes those of excitement and delirium, but more frequently somnolence, which may be increased to deep stupor or coma; (2) fever, usually moderate, but exceptionally very high; (3) disturbed respiration, most frequently deep and rapid; (4) diarrhoeal stools of great variety, no special type being characteristic; (5) very rapid loss of weight; (6) a polymorphonuclear leucocytosis, generally between 20,000 and 30,000; (7) urine containing albumin and casts; (8) the presence of lactose in the urine, if lactose is given in the food; (9) marked general collapse. Associated with these characteristic symptoms there may be almost any others which are found in a severe intestinal condition.

With such symptoms as have been described the usual course is rapidly downward with a fatal termination. If the condition is recognised, however, and properly treated, many cases recover. The essential treatment consists in withholding food of every description and giving water in as large quantities as can be tolerated without vomiting. With a cessation of the most severe symptoms a gradual return to food should be made, the first articles allowed being nitrogenous foods, such as broth, white of egg, beef juice, and buttermilk, or fat-free milk without additional sugar.

**Cholera Infantum.**—This is only one type of acute intestinal intoxication, yet clinically it differs from the others sufficiently to deserve separate consideration. It is not, however, a frequent form. As yet it has not been connected with a specific type of intoxication or infection.



What it is that determines the marked and characteristic symptoms in cholera infantum is entirely unknown. The symptoms are due primarily to the effects of some poison upon the heart, the nerve-centres, and the vaso-motor nerves of the intestines; secondarily to the abstraction of fluid from the various organs and tissues of the body, especially the nerve-centres.

Cholera infantum rarely occurs in an infant previously healthy. As a rule, there is some antecedent intestinal disorder. The development of the choleriform symptoms is usually very rapid, and a child, who perhaps has been regarded as scarcely ill enough to require a physician, may be brought, in the course of five or six hours, to death's door.

Usually there are general symptoms, such as prostration and a steadily rising temperature, for a few hours before the vomiting and purging, or these symptoms may be the first to excite alarm. Vomiting may precede diarrhœa, or both may begin simultaneously. The vomiting is very frequent. First, whatever food is in the stomach is vomited, then serum and mucus, and sometimes there is regurgitation from the small intestine. If vomiting subsides for a time, it is almost sure to begin anew with the taking of food or drink. The stools are frequent, large, and fluid, and may occur once or twice an hour. They are of a pale green, yellow, or brownish colour in the beginning, but as they become more frequent they often lose all colour and are almost entirely serous. The sphincter is sometimes so relaxed that small evacuations occur every few minutes. The first stools are usually acid, later they are neutral, and when serous they are alkaline. In most cases they are odourless; in rare instances they are exceedingly offensive. Microscopically the stools show large numbers of epithelial cells, some leucocytes, and immense numbers of bacteria.

Loss of weight is more rapid than in any other pathological condition in childhood; it may be as much as a pound a day. The fontanel is depressed, and in rare instances there may be overlapping of the cranial bones. The general prostration is great almost from the outset. The face, better, perhaps, than any single symptom, indicates what a profound impression has been made upon the system. The eyes are sunken, the features sharpened, the angles of the mouth drawn down, and a peculiar pallor with an expression of anxiety overspreads the whole countenance, which becomes almost Hippocratic. In the early stages the nervous symptoms are those of irritation. Later, these symptoms give place to dulness, stupor, relaxation, and coma or convulsions.

The temperature, in my experience, has been invariably elevated, and usually in proportion to the severity of the attack. In cases recovering, it has generally been from 102° to 103° F., while in fatal cases it has risen almost at once to 104° or 105° F., and often shortly before death it has reached 106° or even 108° F. Such temperatures may occur

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with a clammy skin and cold extremities, and are discovered only by the thermometer. The pulse is always rapid, and very soon it becomes weak, often irregular, and finally almost imperceptible. The respiration is irregular and frequent, and may be stertorous. The tongue is generally coated, but soon becomes dry and red, and is often protruded. The abdomen is generally soft and sunken. There is almost insatiable thirst. Everything in the shape of fluids, especially water, is drunk with avidity, even though vomited as soon as it is swallowed. Very little urine is passed, sometimes none at all for twenty-four hours; this depends upon the great loss of fluid by the bowels.

In the fatal cases there is hyperpyrexia, a cold, clammy skin, absence of radial pulse, stupor, coma or convulsions, and death. The diarrhœa and vomiting may continue until the end, or both may entirely cease for some hours before it occurs. The patients may pass into a condition resembling the algid stage of epidemic cholera, and die in collapse. In other cases, after the first day of very severe symptoms, the discharges diminish, but the nervous symptoms become specially prominent. There is restlessness and irritability or apathy and stupor. The fontanel is sunken; the eyes are half open and covered with a mucous film; respiration is irregular and superficial, sometimes even Cheyne-Stokes; the pulse is feeble, irregular, or intermittent; the muscles of the neck drawn back; the abdomen retracted. The temperature is not elevated, but normal or subnormal. From this condition recovery may take place or the symptoms may merge into those of ileo-colitis; but much more frequent than either of the foregoing is the fatal termination.

These nervous symptoms are ascribed to cerebral anæmia, cerebral hyperæmia (venous), œdema of the meninges, thrombosis of the cerebral sinuses, and uræmia. Although I have examined the brain in almost all my autopsies upon patients dying from diarrhœal diseases, I have never in such cases seen sinus thrombosis, and but rarely œdema. Cerebral hyperæmia was often met with in cases dying in convulsions, but not with any regularity otherwise. Nor have my observations upon the kidneys confirmed those of Kjellberg, whom most of the writers since his day have quoted, as to the great frequency of nephritis. A scanty, concentrated, and hence irritating urine is the rule, and a small amount of albumin and an occasional hyaline cast not uncommon; but either clinical or pathological evidence of a serious amount of nephritis has been, in my own experience, extremely rare.

We can hardly regard either the renal or the cerebral changes as an explanation of the nervous symptoms of most of these cases; they seem rather to depend upon impeded circulation due to a thickening of the blood, to acute inanition, and general toxæmia.

An infrequent complication of cholera infantum is sclerema. This condition is found associated with muscular contractions, subnormal tem-

perature, and other signs of the most extreme depression. These cases are invariably fatal.

Of the children with true cholera infantum which have come under my notice, fully two-thirds have died.

*Treatment.*—Restricting the term cholera infantum to the class of cases described above, all who have seen much of the disease must admit that the results of treatment are extremely unsatisfactory, and that the most severe cases pursue their course but little, if at all, influenced by the treatment employed.

The best view of the treatment will be gained if we keep in mind that we are treating cases of poisoning; that the toxic materials cause great depression of the heart and the system generally by acting on the nerve-centres, and by paralysing the vaso-motor nerves of the intestine.

The main indications are: (1) to empty the stomach and intestine; (2) to neutralise the effect of the poison upon the heart and nervous system; (3) to supply fluid to the blood to make up for the very great drain of the discharges; (4) to reduce the temperature; (5) to treat special symptoms as they arise.

For the first indication we must rely upon mechanical means—stomach-washing and intestinal irrigation—there is no time to wait for cathartics. For the second, nothing in my hands has proved so useful as the hypodermic use of morphine and atropine. I believe this to be more efficient than any other means of treatment we possess. Morphine is contraindicated where the purging has ceased or is slight, and where there is drowsiness, stupor, or relaxation. The effects of the dose should always be carefully watched; a small dose repeated is better than a single large dose. For a child a year old, not more than gr.  $\frac{1}{6}$  of morphine and gr.  $\frac{1}{16}$  of atropine should be the initial dose. It may be repeated in an hour unless the desired effects are produced: arrest of the vomiting and purging (or at least their diminution), improvement in the heart's action, and in the nervous symptoms.

For the third indication the only thing that can be depended upon is the injection of normal salt solution into the cellular tissue of the abdomen, buttocks, thighs, or back. At least half a pint should be used at a time; it should be injected in several places and repeated in the course of every twelve hours. A very much larger quantity can often be used with advantage. This causes no irritation, and is absorbed with surprising rapidity. The injection is made slowly, and the exact amount introduced at each time measured.

For the reduction of temperature, baths should be used. They may be continued from ten to thirty minutes, and to be efficient, must be used frequently—as often as every hour, if symptoms are threatening. Iced cloths or an ice-cap should be applied to the head. Cold-water injections are a valuable accessory to the treatment by baths. In most cases noth-

ing should be allowed by the mouth except water. Caffein, camphor, and brandy may be used freely. While the use of stimulants is indicated in every case, their effects are disappointing. Taken by the mouth they are almost invariably vomited. If used at all, it should be hypodermically. During the stage of most acute symptoms, to attempt to give food by the mouth is worse than useless. After the stage of violent symptoms has subsided and reaction is established, the subsequent management in respect to feeding and medication should be the same as in the cases considered in the previous chapter. If cerebral symptoms are present, opium is to be avoided. For cold extremities and subnormal temperature, hot mustard baths should be used to establish reaction, mustard paste applied all over the body, and hot-water bags or bottles placed about the patient.

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## CHAPTER VII.

### DISEASES OF THE INTESTINES.—(Continued.)

#### ACUTE ILEO-COLITIS.—DYSENTERY.

(*Entero-colitis; Enteritis; Inflammatory Diarrhæa.*)

THE term *ileo-colitis* is a general one, embracing those forms of intestinal disease in which true inflammatory lesions are present. In the type of cases described in the previous chapter recovery or death takes place before anything more than superficial changes have occurred, while in *ileo-colitis* the pathological process continues until there have been produced marked lesions, often involving all the walls of the intestine. Sometimes it is impossible, by symptoms, to draw a line between them. This is especially true of the cases terminating in follicular ulceration of the colon. In certain other forms of *ileo-colitis* the evidences of a severe intestinal inflammation are often manifest from the very outset. This difference is probably due to a difference in the character of the infection. The extent of the lesions depends much upon the duration of the process.

**Etiology.**—The predisposing causes of *ileo-colitis* are those common to diarrhoeal diseases in general, and have already been considered. Although seen with especial frequency in summer, and in children under two years old, it may affect those of any age, and occurs at all seasons. Epidemics are not uncommon in the early fall months. While usually primary, *ileo-colitis* often follows infectious diseases, especially measles, diphtheria, and broncho-pneumonia. It frequently occurs, in institutions chiefly, as a terminal infection in infants suffering from extreme malnutrition or marasmus. All other forms of intestinal disease are

predisposing causes. The question of contagion is unsettled; if at all communicable, it is feebly so. When it occurs epidemically a common origin seems more probable than that the disease spreads from one patient to another.

The only bacterium that up to the present time has been shown to be capable of producing this form of intestinal disease is the *B. dysenteriae* of Shiga. This organism, or, more properly speaking, this group of closely allied organisms, has now been found in all parts of the world in a sufficient number of cases to establish its etiological connection with ileo-colitis. The *B. dysenteriae* was shown by Shiga, in 1898 and 1899, to be the cause of epidemic dysentery in Japan. In 1900, Flexner established its association with tropical dysentery in the Philippines, and in 1902, Duval and Bassett, pupils of Flexner, demonstrated its presence in a series of cases of diarrhoea in children at Baltimore.

In 412 cases of diarrhoea studied in the summer of 1903 this organism was present in 270. It was almost invariably found in cases showing blood and mucus, or much mucus in the stools. Although usually the *b. dysenteriae* is greatly outnumbered by other organisms, it is not uncommon to find it in pure culture. A number of minor differences have been found in the bacilli from different cases; there are, however, two main groups, the division being made by reason of the difference in reaction with litmus mannite; one group is known as the "true Shiga," or "alkaline" type; the other, as the "Flexner," or "acid" type. The latter has been most frequently found in the diarrhoeal diseases of children in this country, although the true Shiga is occasionally present, and in rare cases they may be associated.

Whether the *b. dysenteriae* is present in normal stools of healthy children is still unsettled. Wollstein at the Babies' Hospital failed to discover its presence in the stools of 56 normal infants. The *b. dysenteriae* has never been found outside the body; we are therefore entirely ignorant both of its habitat and its mode of entry. There are grounds for believing that it appears at times among the saprophytic bacteria of the intestinal contents.

The rôle played by other bacteria, especially the streptococcus, in the production of the deeper lesions of the intestine may be an important one. This appears, however, to be rather in the nature of a secondary invasion.

**Lesions.**—It is surprising that, so far as is known, a single specific cause can excite such a variety of lesions. The nature of the anatomical changes apparently depends upon other factors, such as the intensity of the infection, the local resistance, and still more upon the duration of the disease.

The nature of the lesions in ileo-colitis differs greatly, but their position is quite constant: they affect the lower ileum and the colon.

In about half the cases only the colon is affected. The lesions of the ileum are usually limited to the lower two or three feet.

The frequency with which the different varieties of ileo-colitis were found in eighty-two of my own autopsies was as follows:

|  |          |
|--|----------|
| Follicular ulceration .....                              | 36       |
| Catarrhal inflammation .....                             | 26       |
| Catarrhal inflammation with superficial ulceration ..... | 6        |
| Membranous inflammation .....                            | 14       |
|  | <hr/> 82 |

**ACUTE CATARRHAL ILEO-COLITIS.**—In the milder cases there are changes in the epithelium and infiltration of the mucosa. In the severer cases the submucosa is involved, and the infiltration of the mucosa may be so great as to lead to necrosis and the formation of ulcers.

*Gross Appearances.*—While the lower ileum and the colon are most seriously affected, it is not uncommon to find quite marked changes in a considerable portion of the small intestine, and even in the stomach. In the cases of short duration, the lesions are sometimes more marked in the small intestine than in the colon. The stomach contains undigested food, and mucus which is commonly stained a dark-brown colour. It may be dilated or contracted. The mucous membrane is pale or congested; if the latter, it is usually in patches, and more about the pyloric orifice. The intestinal contents are generally green in colour, and thin. The mucous membrane is often coated with tenacious mucus. The small intestine is distended with gas, the large intestine nearly empty, except the transverse colon. The mucous membrane may appear somewhat swollen. In the small intestine there are occasionally seen swelling and œdema of the villi, so that they project abnormally and give a plush-like appearance. Congestion is a constant feature, and it may be simply upon the folds of the mucous membrane, or about the solitary follicles, or it may be intense and involve the whole intestine for some distance. Small hæmorrhagic areas are often seen here and there, widely scattered. In the most severe cases there are marked thickening and uniform congestion, and the appearance is sometimes much like that seen in membranous inflammation. The solitary follicles throughout the colon are usually swollen, projecting above the mucous membrane and about the size of a pin's head. Peyer's patches may be normal, or they may be swollen and congested, with other evidences of catarrhal inflammation in the surrounding mucous membrane, or, more rarely, they may be involved when the rest of the mucosa appears healthy. The same is true of the lymph nodules of the small intestine. The lymph nodes of the mesentery are usually swollen and acutely congested, but they may appear normal.

*Microscopical Appearances.*—In interpreting the changes found in the mucosa, the same precautions must be observed as previously stated.

There is usually loss of the superficial epithelium and of that lining the tubular glands at their orifices. Upon the surface of the mucosa and within the tubular glands, fine granular matter is seen derived from the broken-down epithelium. The goblet cells are distended with mucus, and do not stain clearly. The lumen of the tubular glands is narrowed from pressure due to the swelling of the lymphoid tissue which separates them, which is partly from œdema, and partly from cell infiltration (Fig. 54). A thick layer of mucus and round cells, adhering closely to the surface, may resemble a pseudo-membrane (Fig. 55). In fatal cases of moderate severity the superficial portion of the mucosa is infiltrated with round cells and crowded with bacteria of many kinds, the depth to which this infiltration extends depending upon the severity and duration of the process. In very severe cases there is found a dense

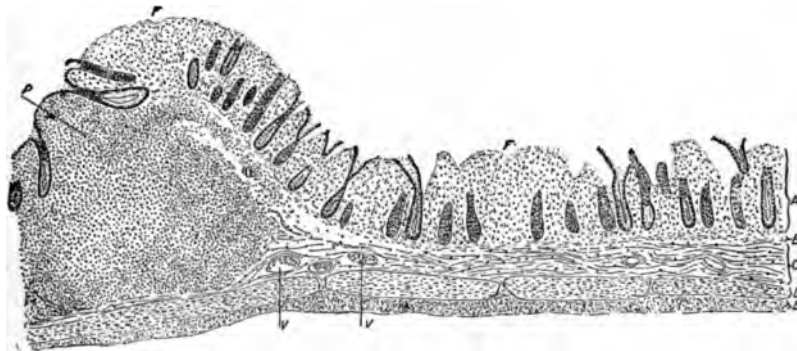
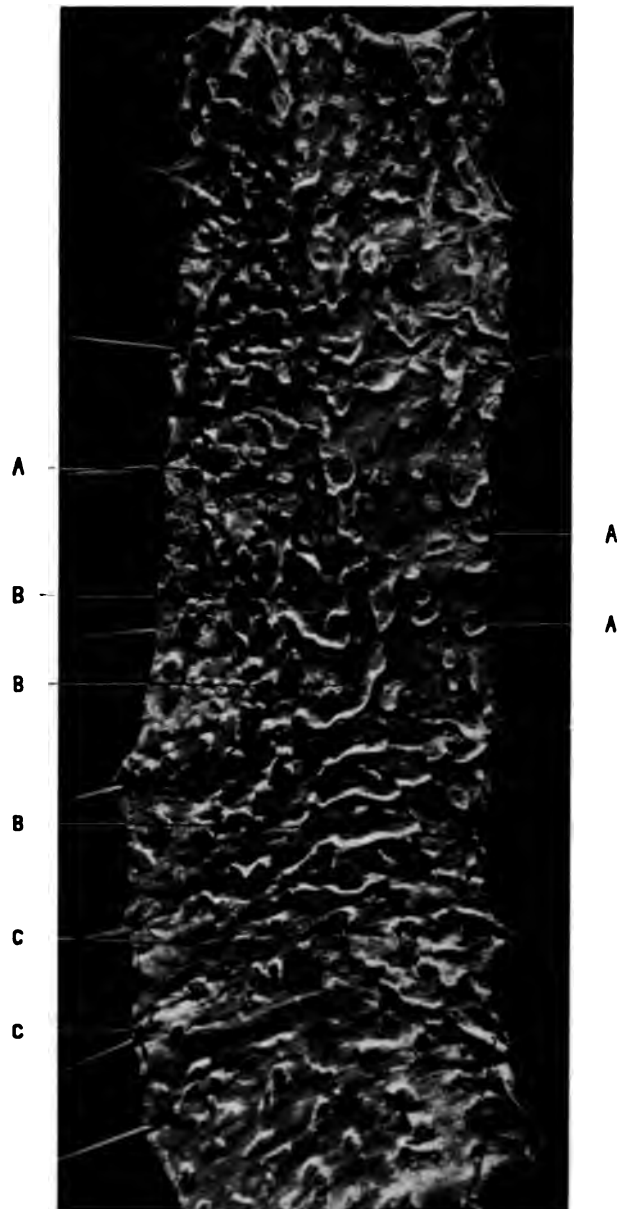


FIG. 54.—ACUTE CATARRHAL INFLAMMATION OF THE ILEUM. At the left is seen the edge of a Peyer's patch (*P*) greatly swollen. The most striking feature of the lesion is the loss of the superficial epithelium, which is shown in all parts of the specimen. The significance of this depends upon the fact that the autopsy was made but two hours after death. At several points, *F, F*, the tubular follicles have loosened and fallen out. The mucosa, *A*, is slightly infiltrated with cells, especially near the Peyer's patch. The submucosa, *C*, and muscular coats, *D, E*, are normal. *V, V*, are small veins. *History*.—Infant, nine months old, previously healthy; sick three days with severe intestinal symptoms; temperature, 103° to 105° F. *Autopsy*.—Acute catarrhal inflammation of ileum and colon; Peyer's patches red and swollen. The specimen is taken from the lower ileum. The superficial character of the lesion is chiefly due to the short duration of the process.

infiltration of the mucosa and of the submucosa also, which in places extends quite to the muscular coat. These cases closely resemble those of the membranous variety, lacking only the exudation of fibrin. The lymph nodules of the colon are swollen to a greater or less degree, chiefly from an increase in the number of lymphoid cells. This swelling may be the most prominent feature of the lesion. If the process is sufficiently prolonged, the lymph nodules may break down and ulcerate. The changes in the lymph nodules of the small intestine and in Peyer's patches are similar to those seen in the colon, but are less marked, and frequently absent altogether. Ulceration in Peyer's patches is extremely rare.

PLATE VIII.



EXTENSIVE SUPERFICIAL ULCERATION OF THE COLON.

Female child nine months old: symptoms of acute ileo-colitis of fifteen days' duration; temperature, 101° to 104·5° F., and from six to eight stools daily—thin, green, and yellow, but no blood.

Extensive ulceration throughout the colon, most marked in descending portion, from which specimen is taken.

A A are small circular ulcers; B B, larger ones from coalescence of several of these; C C, large areas of ulceration, the mucous membrane being almost entirely destroyed.





The small veins and capillaries of the mucosa and submucosa are usually distended with blood; small extravasations are very common, and occasionally larger ones are seen.

Catarrhal inflammation, except in its very severe form, which is not frequent, causes no lesions that can not readily be repaired. The most

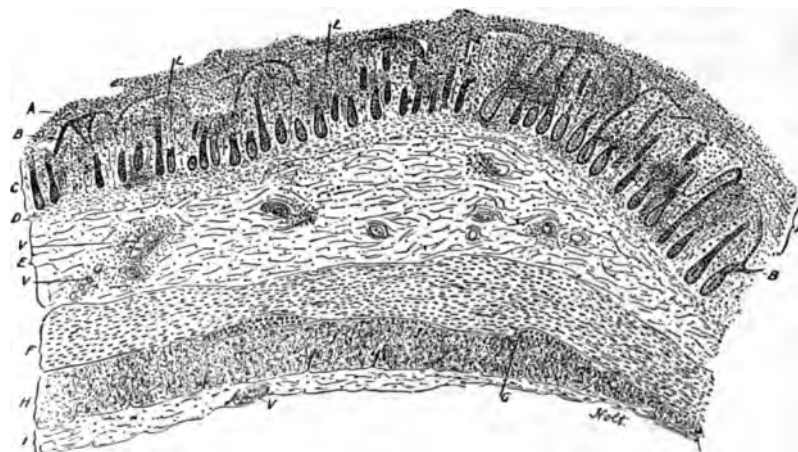


FIG. 55.—ACUTE CATARRHAL INFLAMMATION OF THE ILEUM; SEVERE FORM. The mucosa, C, is everywhere densely infiltrated with round cells, compressing the tubular follicles, and in places, L, L, almost effacing them. Upon the surface of the mucosa is a thick layer of cells and mucus. Beneath this the epithelial arches, B, B, covering the villi can be seen. The lesions are almost entirely of the mucosa. The only changes in the submucosa, E, are groups of cells about the small blood-vessels, V, V. *History*.—Infant six months old; moderate diarrhoea twelve days; severe symptoms with high temperature for six days. There was intense inflammation of the entire colon and lower three feet of the ileum. Intestine greatly congested and thickened. Specimen is from the ileum.

persistent change is usually the swelling of the lymph nodules, which may last a long time, and appears to be an important factor in the tendency to relapses and recurring attacks. If there is a continuance of the exciting cause, or the patient's constitution is feeble, the process may become chronic.

**CATARRHAL INFLAMMATION WITH SUPERFICIAL ULCERATION.**—In the most severe form of catarrhal inflammation which does not prove fatal in the earlier stages, extensive ulceration occasionally takes place; usually these ulcers are seen throughout the entire colon, and occasionally a few are found in the lower ileum. They generally begin in the mucosa overlying the lymph nodules, and while they have a wide superficial area, they do not extend deeper than the mucosa. The small ulcers are circular and usually show at the centre a small granular body—the lymph nodule. The larger ulcers result from the coalescence of several small ones, and are irregular in shape. They may be two or three inches in diameter. Sometimes for a considerable distance a large

part of the mucosa may be destroyed. Often the entire surface presents a worm-eaten appearance (Plate VIII). On microscopical examination there is seen, in the greater part of the ulcer, complete destruction of the mucosa, the submucosa being densely packed with round cells quite to the muscular coat.

**INFLAMMATION OF THE LYMPH NODULES WITH ULCERATION** (*follicular ulceration*).—Follicular ulcers are found at autopsy in about one-third of the cases dying from diarrhoeal diseases. They are rarely seen

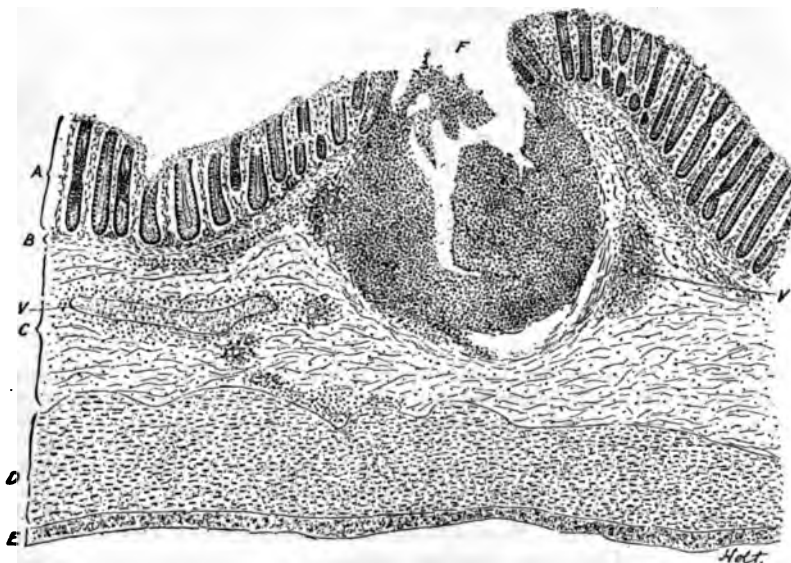
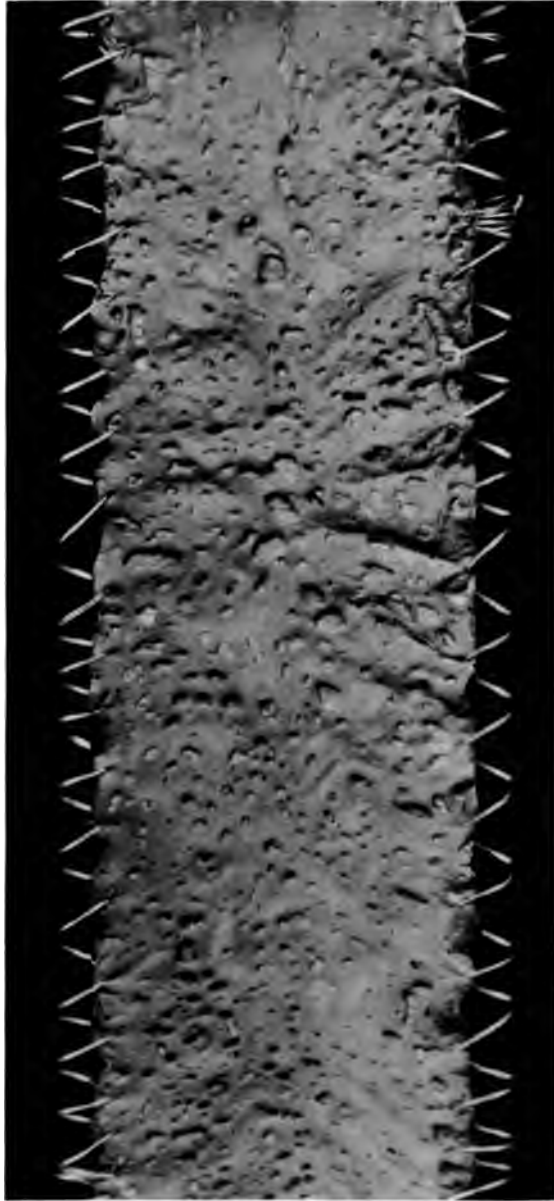


FIG. 56.—LYMPH NODULE OF THE COLON IN THE EARLY STAGE OF ULCERATION—FOLLICULAR ULCER. The nodule, *F*, is much enlarged, and is breaking down and discharging into the intestine. The other changes are not marked. The superficial epithelium is gone; the mucosa, *A*, shows a slight increase of cells, and in the submucosa, *C*, are nests of cells about the small vessels, *V, V*. *History*.—Delicate child, thirteen months old; slight diarrhoea four weeks; severe symptoms five days. The colon was filled with ulcers one-twelfth of an inch in diameter, one of which is shown in the illustration.

in those which have lasted less than a week, and not often before the middle of the second week. The average duration of the disease in these cases is about three weeks.

In thirty-six cases in which follicular ulcers were found at autopsy, they were present in the small intestine alone in but three cases; in the small intestine and in the colon in six cases; in the remaining twenty-seven they were present only in the colon. When in the small intestine they were seen only in the lower ileum. Ulceration was seen a few times in one or two of the nodules of a Peyer's patch. Ulceration of the large intestine involved the whole colon in about half the cases; while in the remainder the process was limited to its lower portion. The deepest and also the largest ulcers were usually in the descending colon and sigmoid flexure.

PLATE IX.



DEEP FOLLICULAR ULCERS OF THE COLON.

A delicate child, fourteen months old, sick twelve days; stools green, yellow, brown, and watery; no blood; temperature, 100° to 101° F.

The small intestine was normal; ulcers throughout colon. The specimen is from descending colon; the ulcers are deep, and most of them extend to the muscular coat. (For microscopical appearance, see Fig. 68.)



In the early stage these ulcers appear as tiny excavations at the summit of the prominent lymph nodules. Later, the whole nodule may be destroyed, and a small round ulcer is formed from one-twelfth to one-fourth of an inch in diameter (Plate IX). These are quite deep and have overhanging edges; when closely set they give the intestine a sieve-like appearance. By the coalescence of several of them, larger ulcers may form which are an inch or more in diameter. At the bottom of these larger ones the transverse striæ of the circular muscular coat are often plainly seen. I have never known them to cause perforation.

*Microscopical Appearances.*—The lymph nodules are swollen, principally from the accumulation within them of round cells. This is followed by softening, which usually begins at the summit of the nodule and extends downward; the reticulum breaks down, and the cellular contents escape into the intestine (Fig. 56). Softening may begin at the centre of the nodule, which ruptures like an abscess. The destruc-

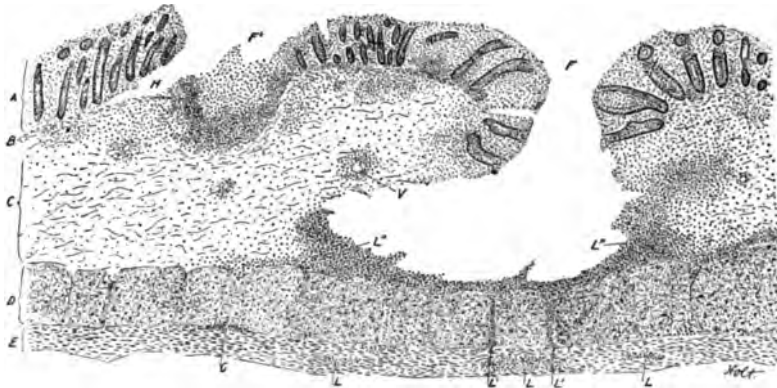


FIG. 57.—DEEP FOLLICULAR ULCER OF THE COLON. A deep ulcer is shown at *F*, a smaller one at *F'*. The separation of the mucosa at *H* is accidental. There is no trace of the lymph nodule from which the large ulcer had its origin. The destructive process has extended laterally in the submucosa, *C*, and the mucosa, *A*, is falling in to fill up the space. In the vicinity of the ulcers, the submucosa is densely infiltrated with round cells, *L'*, *L''*, which also are seen in the lymph spaces between the bundles of circular muscular fibres, *L'*, *L''*, and some are seen in the longitudinal muscular coat, *L*, *L'*. *History.*—Thirteen months old, delicate; continuous diarrhoeal symptoms for three weeks. Ulcers found throughout the colon, the largest, one-half an inch in diameter. The illustration shows one of the small ones like those in Plate IX.

tion of the whole nodule leaves a cavity, which is the follicular ulcer. At first the ulcer corresponds in size to the nodule, but infiltration of the adjacent tissue soon takes place, which may become necrotic. In this way the ulcer extends chiefly in the submucous coat. The lesion is never limited to the lymph nodules; but the extent of the other changes found depends upon the severity and the duration of the process. In cases dying after an illness of a week or ten days, we usually find only moderate changes in the mucosa, and in the submucosa a slight infiltra-

tion of round cells, especially about the small blood-vessels (Fig. 56, V, V). In those which have lasted three or four weeks the ulcers are deeper, and all the structures of the intestine in their neighbourhood are usually involved (Fig. 57). The mucosa is densely packed with round cells, as are also all the tissues in the vicinity of the ulcers; even the muscular coat may be infiltrated. The ulcers, however, rarely extend deeper than the circular layer.

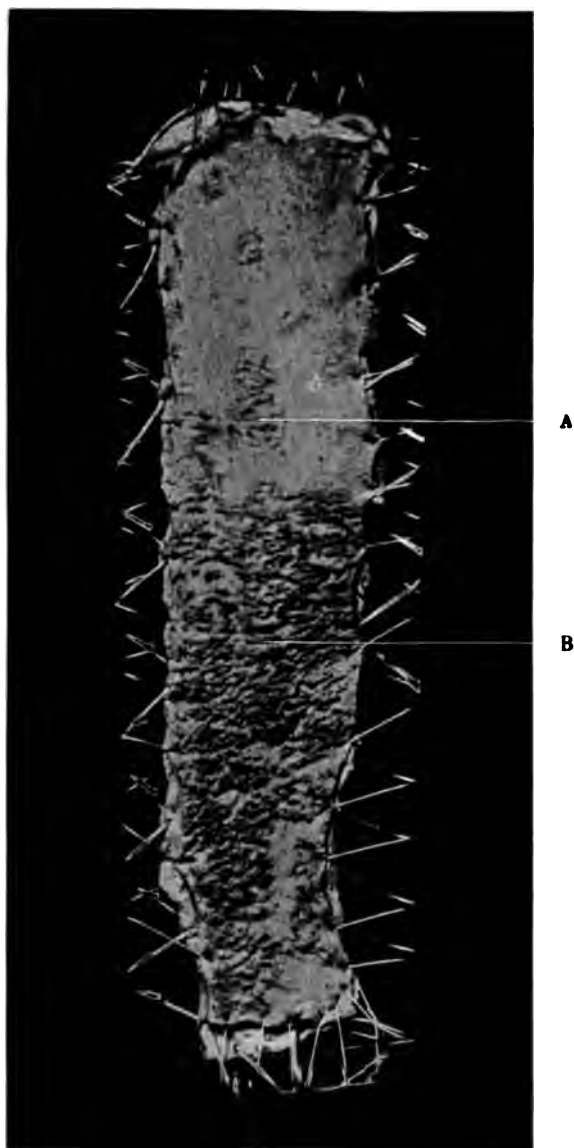
Follicular ulceration of the intestine in infancy usually terminates fatally if the process is an extensive one. In less severe cases recovery may take place, the ulcers healing by granulation and cicatrization in the course of from four to eight weeks.

**ACUTE MEMBRANOUS ILEO-COLITIS.**—This is the most severe form of intestinal inflammation seen among children. The process differs quite materially from that described as occurring among adults. In only one of my own cases was it associated with membranous inflammation of any other mucous membrane, in that case with membranous gastritis. The most frequent type of membranous colitis is that with severe acute symptoms, both constitutional and local, with a duration of from six to fourteen days. In young infants its symptoms and course are very irregular, and it may be found at autopsy when no serious intestinal lesion has been suspected.

*Gross Appearances.*—There is visible to the naked eye usually very little pseudo-membrane and no deep sloughing. The lesion affects the last two or three feet of the ileum and the entire colon, sometimes only the colon. It is exceedingly rare to meet with any marked lesions higher in the small intestine. The most marked changes are near the ileo-cæcal valve or in the sigmoid flexure and the rectum. In the ileum they may be quite as severe as in the colon (Plate X). The intestinal wall is firm and stiff, and is two or three times its normal thickness. It is not thrown into deep folds, as is the healthy intestine when empty. It is very rare to find false membrane that can be stripped off in patches of any considerable size. When membrane exists, the colour is a yellowish or grayish green, and the surface is often fissured, giving a lobulated appearance. In the parts where no pseudo-membrane can be seen, the surface is usually of an intense red colour and is rough and granular, in striking contrast to the normal glistening appearance. Here and there small extravasations of blood may be seen. In the regions most affected, the normal structures of the mucous membrane—the villi, Peyer's patches, and solitary follicles—can not be distinguished. In a single instance I found an exudation of fibrin on the peritoneal surface of the intestine for a short distance. Except in the lower ileum the small intestine shows no constant changes, and none are usually found in the stomach.

*Microscopical Changes.*—These (Fig. 58) are much more uniform than the gross appearances. The most characteristic feature is the exu-

**PLATE X.**



**MEMBRANOUS INFLAMMATION OF THE ILEUM.**

A delicate child, eleven months old; mild diarrhoea for two weeks without fever; acute severe symptoms for twelve days; temperature, 100° to 102.5° F.; green and mucous stools; no blood.

The lesions involved the last foot of ileum and entire colon. Specimen is from lower ileum, and shows the abrupt termination of the lesion; the upper part shows normal small intestine; A is a Peyer's patch; B is the inflamed part of the intestine; it has a rough granular appearance and is much thickened.





dation of fibrin, which forms a distinct pseudo-membrane upon the surface of the intestine; it may infiltrate the mucosa, and even the sub-mucosa. Fibrin is seen under the microscope in parts of the specimen, which to the naked eye show no distinct pseudo-membrane, but only a granular appearance. In rare cases a fibrinous exudation may be found upon the peritoneal covering of the intestine. The pseudo-membrane is made up of a fibrinous network containing small round cells, some red

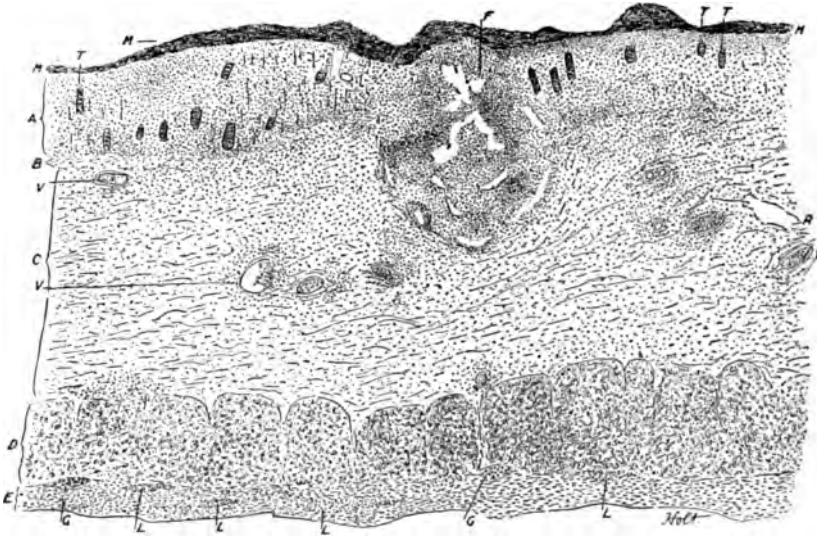


FIG. 58.—MEMBRANOUS INFLAMMATION OF THE COLON. The intestine is covered with a pseudo-membrane, *M*, which is composed chiefly of granular fibrin; the mucosa, *A*, is densely packed with round cells, and the tubular follicles have almost disappeared, traces only being left at *T, T*. The submucosa, *C*, is greatly thickened, partly from cells, but chiefly from fibrin, which with a high power is seen to be everywhere in this coat, as well as the mucosa. Nests of cells are seen in the muscular coats at *L, L*. At *F* is a lymph nodule covered by pseudo-membrane, but breaking down at its centre. *V, V*, are small blood-vessels with nests of cells about them. *History*.—Fourteen months old; ill nine days; temperature 101° to 105° F.; all stools containing blood. Lesions found throughout colon and in lower ileum. Intestine greatly thickened. Specimen is from ascending colon, where lesion was especially severe.

blood-cells, and numerous bacteria. The mucosa, and usually the sub-mucosa, are densely infiltrated with small round cells, which in places may be so numerous as to efface the normal elements of the intestine. The tubular follicles are in some places quite destroyed, not a vestige of them remaining. In other places they are compressed and distorted by the accumulation of cells. The great thickening of the intestine is due partly to the cell infiltration, partly to the fibrinous exudation, and partly to œdema. All the blood-vessels, both in the mucosa and sub-mucosa, are gorged with blood, and many small extravasations are seen.

A necrotic process with the formation of deep ulcers I have never seen associated with membranous colitis.

**ASSOCIATED LESIONS OF ILEO-COLITIS.**—The most important one is broncho-pneumonia. It is found in quite a large proportion of the protracted cases, and not infrequently it is the cause of death. I think it is seldom due to an infection from the intestine, although such a thing is possible in septicæmic cases. It occurs rather as it does in any other protracted exhausting disease. In a study of sixty cases, Spiegelberg did not find bacteria in the pulmonary capillaries, and he regards infection through the blood as not yet proved. Pulmonary tuberculosis is not infrequently met with in hospital cases, having no relation to the intestinal disease. Peritonitis is infrequent. I have met with it but once or twice, and then it was localised and of the plastic variety. Inflammations of the other serous membranes—pleurisy, pericarditis, and meningitis—are all very rare.

The renal lesions of ileo-colitis have been the subject of considerable discussion, some observers holding that nephritis is a frequent complication of the severer forms of diarrhœa, while others have held it to be rare. The lesions I have usually found in my own cases coincide with those described by others, and consist in marked degeneration of the epithelium of the tubes with but few glomerular or interstitial changes. In three or four instances only have I found well-marked lesions of acute diffuse nephritis at autopsy, or seen its symptoms clinically. I believe it to be a very infrequent though sometimes a most serious complication. The lesions mentioned as usually present are properly classed as acute degeneration rather than as inflammation of the kidney. Its causes are chiefly the irritation of toxins, intensified no doubt by the concentration of the urine. Degenerative changes may be found also in the heart muscle, the liver, spleen, and even in the central nervous system.

Considerable attention has been given to a study of the blood in intestinal inflammations, to determine how frequently and in what circumstances a general blood infection (septicæmia) from the intestines occurs. In the great majority of the cases studied under proper precautions the blood is sterile.

**Symptoms.**—(1) *Catarrhal Cases of Moderate Severity.*—The onset is usually sudden, often with vomiting, and for twelve, sometimes twenty-four hours the symptoms may be those of acute indigestion: vomiting, pain, fever, and frequent, thin, green or yellow stools, which are partly faecal and contain undigested food. Later the discharges contain blood and mucus, are often preceded by pain and accompanied by tenesmus. The stools are very frequent, often every half hour, and proportionately small, sometimes less than a tablespoonful being found upon the napkin after severe straining efforts. The mucus may be clear and jelly-

like, or it may be mixed with fæcal matter. Blood is seen in some cases in almost every stool, but rarely in clots, usually streaking the mucus. These stools are almost odourless. After two or three days the blood usually disappears, or is seen only as traces in an occasional stool; but mucus is still present in large quantities. The colour of the discharges now becomes dark brown or brownish-green. Prolapsus ani is frequent, and may occur with nearly every stool. Abdominal pain is present, and is often quite intense just before the stool; and frequently there is tenderness along the colon. For the first twenty-four hours the temperature is usually high, from  $102^{\circ}$  to  $104^{\circ}$  F. During the greater part of

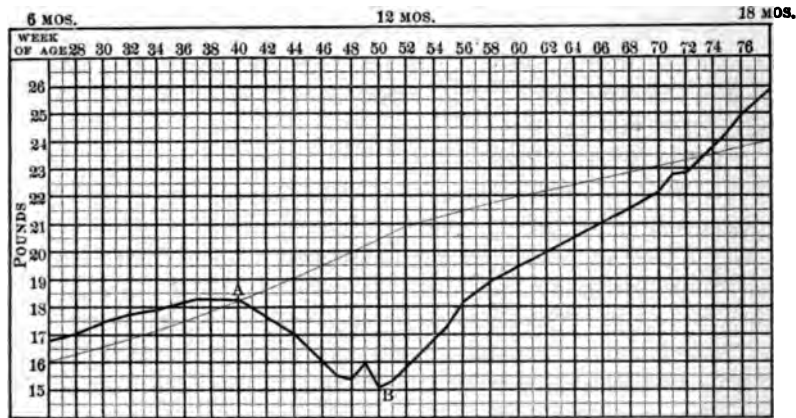


FIG. 59.—WEIGHT CURVE SHOWING LOSS FROM ILEO-COLITIS. Well-nourished infant; attack of measles at A (fortieth week), followed by ileo-colitis, which though not severe continued with exacerbations during September and October. At B all symptoms had disappeared except occasional mucus in the stools. Rapid improvement from this time, which was continued during the winter, the child being sent to a warm climate; it was, however, five and a half months before the weight reached the normal average line.

the attack it ranges from  $99^{\circ}$  to  $102^{\circ}$  F. There is considerable prostration; the loss in weight is usually marked and continuous; appetite is lost; the tongue is coated and the general appearance of the children indicates serious illness, although no really grave symptoms are present. Convalescence is always slow, and it may be months before the lost weight is regained (Fig. 59).

In the milder cases the symptoms point to inflammation of the lower part of the colon only. The constitutional symptoms are not at all marked. The temperature may not be above  $101^{\circ}$  F.; the tongue may remain clean and the appetite good; the child may be bright and active, and hardly seem at all ill, and yet have from six to eight small mucous and bloody stools a day.

The duration of the acute symptoms is usually about a week, and yet in such cases, even though the child was previously in good condition

and properly treated, recovery is slow. The first symptom of improvement is generally the disappearance of blood from the stools, which at the same time become less frequent, and the pain and tenesmus cease. Gradually the stools assume more of a faecal character, but mucus is likely to persist for two or three weeks; it may be seen in all stools, or only occasionally. In some cases both the mucus and blood disappear and the stools become thin, brown, or green, like those of an ordinary diarrhoea. Although the early stage of very acute symptoms may last but a few days, if there is a continuance for three or four weeks of the brown, mucous stools, with emaciation and slight fever, ulceration is probably present. This is likely to occur if the child is in poor condition, if its surroundings are bad, or if it is improperly treated at the outset. Relapses are readily excited, but cases like the above are rarely fatal except in delicate infants. This is the most common form of ileo-colitis which terminates in recovery.

(2) *The Severe Catarrhal Form.*—This form of ileo-colitis, like that just described, is usually primary. The symptoms closely resemble those of the membranous variety, and a diagnosis from it is to be made only by the absence of pseudo-membrane from the stools. The most rapid case I have seen lasted only three days, but the usual duration is from one to two weeks. The temperature is steadily high; the stools continue very frequent and generally contain blood; there is great prostration, dry tongue, sordes on the lips and teeth, and prominent nervous symptoms. Death usually occurs from exhaustion and profound sepsis while the acute symptoms are at their height. If the patient survives this stage, the case may drag on for four or five weeks, very much like one of follicular ulceration, and then terminate in recovery or in death from slow asthenia, broncho-pneumonia, or from an acute exacerbation of the intestinal symptoms. The autopsy in such cases usually reveals the presence of artificial ulcers. If recovery is to be the outcome, after the symptoms have been nearly stationary for a long time, there is seen a gradual improvement first in the general and then in the local conditions. Convalescence is very slow, often interrupted by relapses, and it may be months before the patient is quite well. In some cases the child never regains his former vigour.

(3) *Follicular Ulceration—Ulcerative Inflammation of the Nodules.*—Follicular ulceration is often preceded by other forms of intestinal disease. It is not very frequently met with in infants under six months of age. The great majority of those affected are in poor condition at the time of the attack.

To understand the symptoms of these cases, it must be remembered that follicular ulceration is a terminal process following other forms of diarrhoea. It may be preceded by one or more acute attacks, or by a protracted subacute attack. On account of the feeble resistance of the

child or the continuance of the exciting cause, the pathological process gradually extends to the lymph nodules of the intestine, chiefly the colon, which, as already described, pass successively through the stages of swelling, softening, and ulceration. The onset of the illness may therefore be abrupt, with vomiting and high fever; or gradual, without vomiting and with very little fever. The patient may be ill for a week before the exact type which the disease is assuming can be positively determined. It is not possible to mark the transition from acute gastro-enteric intoxication to follicular ileo-colitis. Usually the latter may be assumed to exist whenever, after a very acute onset, there is a continued temperature, and when the stools habitually contain large quantities of mucus without blood.

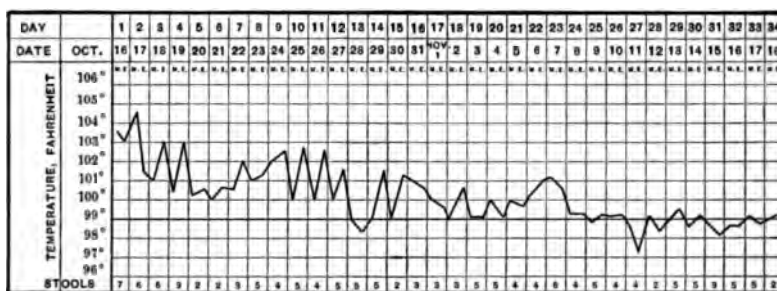


FIG. 60.—TEMPERATURE CHART OF ILEO-COLITIS, FATAL ON THIRTY-FOURTH DAY. Autopsy showed follicular ulcers throughout the colon.

Vomiting is not a feature of these cases; but it is often present at the onset. Throughout the attack it is easily excited by injudicious feeding or medication. The temperature is seldom high, except at first; its usual range is from 99° to 101° F.; toward the close, even of fatal cases, it may be scarcely above the normal. The accompanying chart (Fig. 60) is a very good illustration of the course of the temperature in cases beginning abruptly and ending fatally.

The stools are seldom very frequent, the number being from four to eight a day. The most constant feature is the presence of mucus, which is mixed with the stools and usually abundant. Blood is not generally present, and a large amount of blood is extremely rare. It was absent entirely in more than half of my cases in which the diagnosis was confirmed by autopsy. A small quantity of blood early in the attack is not uncommon, depending here upon congestion. Large hæmorrhages from ulcers I have never seen. The colour of the stools is most frequently dark green or brown. Fluid stools are seen only during exacerbations. The odour is usually offensive, particularly in protracted cases. The microscope shows epithelial cells in great numbers, and very often an abundance of small round cells, which may be looked upon as the most constant sign of ulceration.

The failure in nutrition and steady loss in weight are very constant in these cases. As emaciation goes on, the skin hangs in loose folds on the thighs; it becomes dry and scaly and loses its elasticity, and occasionally small petechial spots are seen upon the abdomen. The skin over the buttocks becomes excoriated, and bed-sores form over the heels, the sacrum, or the occiput. The abdomen may be moderately distended, or it may be relaxed and soft. Tenderness is not usually present. The appetite is lost, and in most cases great difficulty is experienced in getting children to take a proper amount of nourishment. Continued aversion to food is an unfavourable symptom. Occasionally, when there is fever, fluids are taken eagerly. A returning appetite is always an encouraging sign. The mouth is often dry, the tongue coated, sometimes dry and brown; there may be sordes upon the lips and teeth. Superficial ulcers form upon the mucous membrane of the mouth, and often thrush is seen. The urine is usually diminished, high-coloured, and loaded with urates. Albumin and casts are rarely present. In only two or three cases have I seen nephritis severe enough to be a factor in the result. Tenesmus and prolapsus ani are uncommon.

The average duration of the fatal cases is about three weeks; their course is often marked by exacerbations and remissions. If recovery takes place, convalescence is always very slow and relapses are easily excited.

Very few of these cases recover completely. Even those who survive the primary illness are likely to suffer from intestinal symptoms for many months. Fatal relapses are often brought on by injudicious feeding when the children are apparently almost well. The general health is usually so undermined that the patients continue to suffer from all the symptoms of malnutrition, and ultimately succumb to an attack of some intercurrent acute disease.

The diagnosis of ulceration is to be made from the case as a whole rather than from any special symptoms. If a delicate infant, who has previously been prone to diarrhœal attacks, has green mucous stools with low fever, and these symptoms continue with unabated severity for ten or twelve days, ulceration is probable. If such symptoms continue for three or four weeks with steadily failing strength and loss of weight, the diagnosis is almost certain. If, on the contrary, after three or four days of acute symptoms there is improvement in the stools and occasionally some which are quite fœcal in character, even though it may be a week or more before the mucus disappears, we may be quite certain that no ulcers have formed.

(4) *The Membranous Form.*—This is the gravest form of inflammation of the intestines seen in children, and its symptoms are more often obscure than are those of any other variety. This is particularly true when it affects young infants. There may be at the onset and through-

out the course of the disease severe local and constitutional symptoms; or with well-marked constitutional symptoms, the local symptoms may be slight or of very doubtful character, so that it is often mistaken for some other disease.

In the first form it closely resembles the most severe cases of catarrhal inflammation. The disease begins abruptly, with vomiting, high temperature, and several large, fluid stools. The vomiting does not often continue after the first twenty-four hours. The temperature is at first from 102° to 105° F., and its course may be steadily high (Fig. 61), or remittent. The abdomen is often tender and sometimes swollen. There is severe pain, and at times tenesmus, with prolapse of the rectum. This is intensely congested, and sometimes shows patches of pseudo-membrane upon its surface, thus establishing the diagnosis.

The stools often resemble those of the catarrhal variety, except that blood is more constantly present and usually more abundant, but the only positive point of difference is the presence of shreds or flakes of pseudo-membrane. If the stools are thoroughly washed with water these may be seen as small gray opaque masses, which are then easily distinguished from the transparent mucus. Large shreds of membrane are seldom seen in children. Both blood and mucus sometimes disappear from the stools, which may consist only of dirty water. Under the microscope there may be seen epithelial cells, red blood-cells, and round cells in great numbers.

The presence of cerebral symptoms in these cases of membranous ileo-colitis may lead to great obscurity in the diagnosis. This is most frequently true at the onset. There may be high temperature, great prostration, vomiting, stupor, delirium, and even convulsions; and such symptoms may for two or three days completely mask the intestinal condition. As the case progresses, however, the intestinal symptoms come more and more into prominence, and the cerebral symptoms usually subside. But sometimes this is not the case. I once saw a case closely watched for two weeks by three physicians of large experience, who were agreed in the diagnosis of a cerebral lesion, but not as to its nature, which showed at autopsy only the lesions of membranous colitis. There was a continuous but irregular fever, stupor, retracted abdomen, opisthotonus, unequal pupils, and at times irregular respiration. Two or three days before death the first blood appeared in the stools, and at

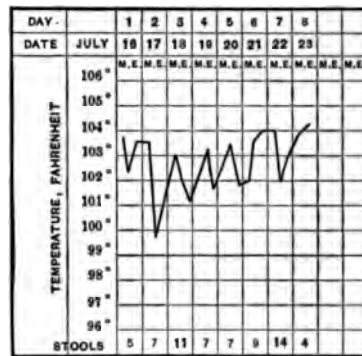


FIG. 61.—TEMPERATURE CHART OF MEMBRANOUS COLITIS; FATAL.



the same time, during extensive rectal prolapse, a false membrane was seen.

Membranous colitis is also obscure when it affects young infants. Every year a number of these cases are seen at the Babies' Hospital. The prominent symptoms are: rather high, continuous temperature, usually ranging between 101° and 104° F., but following no distinct curve (Fig. 62); wasting, which is not rapid but progressive; frequent

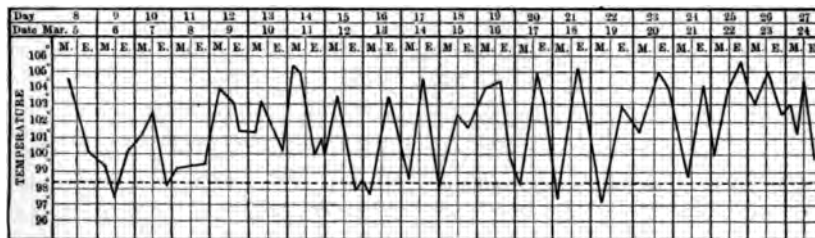


FIG. 62.—TEMPERATURE CHART OF MEMBRANOUS COLITIS. Infant fourteen months old, Babies' Hospital. Symptoms for the first two weeks obscure, suggesting first pneumonia, afterward meningitis. Intestinal symptoms for the last two weeks only, never very severe; stools four to six daily, generally green, thin, with much mucus at times, and once or twice traces of blood. Culture four days before death showed streptococci and colon bacilli. Autopsy: No lesion of importance except membranous colitis involving entire colon; a slight catarrhal enteritis.

stools, which have no constant or striking characteristics. They are usually thin, yellow or greenish in colour, often containing no mucus or blood. Occasionally for a day the stools may be almost normal in appearance. In number they average five or six a day, but often for days only two or three. Outside of a hospital where autopsies are regularly made these cases are usually overlooked and considered as obscure pneumonia, tuberculosis, septicæmia, typhoid, etc.

The duration of membranous ileo-colitis is usually from one to three weeks. Death takes place from sepsis, exhaustion, or from complications. It is probable that almost every case of the severity described terminates fatally when it occurs in an infant. In older children the prognosis is much better as to life, but in them the acute attack may be followed by the chronic form of the disease.

**Diagnosis.**—Ileo-colitis is to be distinguished chiefly from typhoid fever, intussusception, and meningitis. Typhoid is distinguished by the slower invasion, more constant temperature, enlargement of the spleen, tympanites, and most of all by the Widal reaction and the eruption. Acute colitis should not be confounded with intussusception; yet the records of intussusception show that a very large proportion of the cases were regarded in the beginning as cases of dysentery. In intussusception, although we have a sudden onset with acute pain, tenesmus, vomiting, and marked prostration, there is rarely fever. The later symptoms

—absolute constipation, tumour, stercoraceous vomiting, and collapse—have nothing in common with colitis. The membranous form may be confounded with meningitis, and in some cases a differential diagnosis is impossible except by lumbar puncture. Marked diarrhoea, even though the stools are not characteristic, should always make one doubt meningitis.

A diagnosis between the different varieties of ileo-colitis is not always possible. Follicular ulceration is distinguished by its lower temperature, rather subacute course, infrequency of blood in the stools, and by the fact that it is usually preceded by diarrhoeal attacks which are often prolonged.

In the catarrhal form, the symptoms of an acute inflammation of the colon are usually manifest from the outset—bloody stools, pain, tenderness, tenesmus, and fever. In the membranous variety such symptoms are sometimes seen; but, as a rule, the local symptoms are less pronounced, while the constitutional symptoms, especially those relating to the nervous system, are usually marked. The course is usually shorter and more intense than in the other forms.

An agglutination reaction of the *B. dysenteriae* with the serum of affected children is usually present. But for general use in diagnosis this is not of great assistance. It is subject to considerable variation. Moreover, it is seldom present until the end of the first week of the disease, by which time the nature of the attack is evident by clinical symptoms. Agglutination in the higher dilutions is seen only with the particular type of organism with which the infant is infected.

**Prognosis.**—The younger the patient the worse the outlook. The prognosis is rendered unfavourable by extreme summer heat and by prolonged previous attacks of intestinal disturbance. The outlook is worse in secondary than in primary cases. In a given case bad prognostic symptoms are: continuous high temperature, the persistence of much blood in the stools, and severe nervous symptoms. The prognosis is always worse in institutions than in private practice.

**Prophylaxis.**—What has been said regarding general prophylaxis in the previous chapter, applies equally well to cases of ileo-colitis.

Special emphasis should be placed upon the necessity of energetic early treatment of all the milder forms of diarrhoea, and particularly the cases of acute intestinal indigestion and intoxication, in order that the process may be arrested before serious anatomical changes have taken place. Equal stress should be laid upon the importance of prompt and intelligent treatment at the very beginning of the cases with a sudden onset.

**Hygienic Treatment.**—The general plan recommended in the previous chapter should be followed here. A change of air is desirable for most cases as soon as the acute inflammatory symptoms have subsided.

In the protracted cases which drag on a subacute course, this change will often do more than anything else. Plenty of fresh air is necessary in all cases. The indications for bathing are the same as in other cases of acute diarrhoea. It is undesirable to crowd these patients in institutions, as they always do better when separated.

The diet during the acute stage should be the same as in other forms of acute diarrhoea. In the protracted cases the diet presents great difficulties, as the children have little or no appetite, and soon come to refuse everything in the shape of food that is offered. In infancy, in the early stage only, barley or rice water should be given. In the later stage the articles which are most to be depended upon are skimmed milk, which has been sterilised, buttermilk which should be diluted according to the conditions present, and animal broths. Especially to be avoided, not only in the acute stage but during convalescence, are cream, all top-milk mixtures, and also the malted foods. Infants, when very ill, are much more likely to take too little than too much food. A careful record should be kept of the amount actually taken in each twenty-four hours. In no case should food be given oftener than every three hours, and usually the intervals should be longer, water and stimulants being allowed between the feedings. In older children the diet during the acute stage should be much the same as in infants. At a later period, rare scraped beef, kumyss, buttermilk, skimmed milk, and zoolak will be found useful, and during convalescence, eggs, boiled milk, or milk gruels made with rice or barley. Special care should be given to the diet for a long time. For months after an acute attack the intestines are very easily deranged. Relapses are excited by changes in the temperature, by great fatigue or exhaustion, but most of all by improper feeding. Especially in older children should such articles as cream, oatmeal, potatoes, corn, tomatoes, green vegetables, and all fruits be withheld for a long time. I have seen a single peach, given to a child two years old, excite a dangerous relapse, and a few raisins a fatal one.

**Medicinal and Mechanical Treatment.**—Cases, the early stage of which is marked by vomiting and thin diarrhoeal stools, are to be managed at the outset according to the plan outlined in the previous chapter, viz., free purgation, irrigation of the colon, and stopping all food. When the symptoms of acute inflammation are evident from the outset, as shown by the frequent bloody and mucous stools with tenesmus and pain, the measures to be depended upon are castor oil or saline cathartics, irrigation of the colon, and later opium and bismuth by the mouth. Castor oil should be administered in a full dose at the outset—one drachm at six months, two drachms at one year, and half an ounce at four years. Its primary effect is to clear the intestines, and its secondary effect is soothing. The salines may be used as described in the

previous chapter. If the stomach is at all irritable, calomel, one-fourth grain every half-hour for five or six doses, may be substituted. Opium is usually required on account of the pain, tenesmus, and great frequency of stools. The dose should be regulated by the severity of these symptoms. The deodorised tincture and paregoric are, I think, preferable to other preparations. Repeated small doses are better than a single large dose. It is very important that opium should be withheld for at least twelve hours after the initial purgative.

As the pathological process is principally in the colon, and most severe in the lower half of the colon, it can often be much more effectively treated by injections than by drugs given by the mouth. Irrigation of the colon is one of our most valuable means of treatment in these cases. For general purposes a saline solution at 100° to 104° F. should be employed. One or two quarts should be given at one time; it should be injected high into the colon through a rectal tube, and early in the disease repeated at least twice a day. When the tenesmus is very great and blood abundant, small injections of either hot water (106° to 110° F.) or ice water may be used, and later astringent injections.

The most useful astringent is tannic acid of which one drachm may be added to a pint of hot water. Whether injections are to be used regularly or not will depend much upon the patient. If they are well borne, they may be given once or twice a day during the attack; but if at every attempt to give them the child struggles, screams, and resists, they may do more harm than good. Complete rest is a very important part of the treatment.

For cases not influenced by the measures mentioned, or those not seen at the outset, bismuth should be tried, but it is of no use whatever unless large doses are administered. From two to four drachms of the subcarbonate should be given in twenty-four hours to a child two years old, and proportionate doses to older children. This may be suspended in mucilage. Tenesmus and pain are sometimes relieved by the injection of three or four ounces of a starch solution to which from five to ten drops of laudanum are added. Severe tenesmus, when not controlled thus, and when associated with prolapsus ani, is sometimes immediately relieved by a suppository containing cocaine. Not more than one-fourth grain should be used for a child of three years.

Although a serum has been produced which protects animals against inoculation with the *B. dysenteriae*, its use in the treatment of the various forms of ileo-colitis in children has not been followed by any very striking benefit.

Alcoholic stimulants are needed in many cases. They are indicated by a weak pulse, cold extremities, and great general prostration, no matter at what stage in the disease these symptoms are seen. Brandy is usually to be preferred. Generally not more than fifteen or twenty

drops every three hours are needed for an infant one year old. Brandy should always be well diluted.

In cases where symptoms have lasted two or three weeks, and the active ones have subsided, where the temperature is scarcely above 100° F., and the stools reduced to four or five a day, it is wise to stop all medication and attend only to the feeding, with irrigation of the colon every two or three days. One is often surprised at this stage to find that patients do better without drugs than with them. The prevailing tendency is to overdose cases of this type. Careful attention to diet, judicious stimulation, occasional irrigation of the bowel, with change of air, will do much more than any amount of medication. During convalescence general tonics are required, such as arsenic, iron, nux vomica, and wine.

#### CHRONIC ILEO-COLITIS.

The severe forms of chronic ileo-colitis follow acute ileo-colitis, usually the catarrhal or follicular form, as the membranous is so severe that the patients rarely survive the acute stage. There may be only a chronic catarrhal inflammation of the mucous membrane, or ulcers may be present. The milder forms are usually the result of chronic intestinal indigestion.

**Lesions.**—*Catarrhal Form.*—In its milder form it is fairly common, but in its severe form it is exceedingly rare. There may be changes in a large part of the small intestine and in the stomach, as well as in the lower ileum and colon.

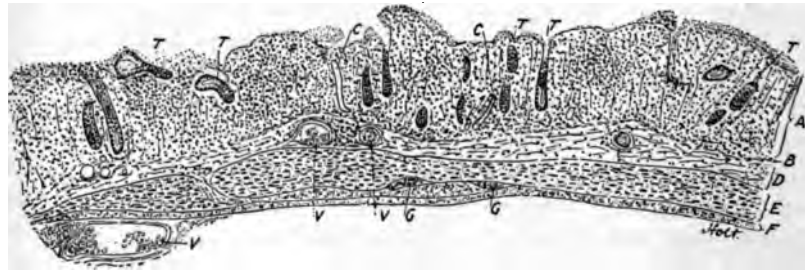


FIG. 63.—CHRONIC CATARRHAL INFLAMMATION OF THE ILEUM. The lesions affect the mucosa, A, almost exclusively. It is somewhat thickened; there is extensive destruction of the tubular follicles, remains being seen at T, T; there is a great increase in the cells, and some new connective tissue in the mucosa. Large new blood-vessels are seen at C, C. *History.*—Delicate child, thirteen months old; diarrhoeal symptoms for four months; during the first two weeks there was high fever; at death weighed eight pounds. The gross changes at the autopsy were very slight. The section is from the middle ileum.

The gross appearance of the intestine often differs very little from the normal. The mucous membrane is usually of a dull gray or slate colour. Pigmentation may occur as striæ in the mucous membrane, but

more frequently it is limited to Peyer's patches and the solitary lymph nodules; these, as well as the mesenteric lymph nodes, are generally swollen.

The microscopical changes are usually marked. The lesion is chiefly one of the mucosa (Fig. 63). The important features are a disappearance of very many of the tubular glands, and, in the small intestine, of the villi also. There is a very marked cell proliferation in the adenoid tissue of the mucosa, and if the disease has existed long enough there may be a production of new connective tissue. The solitary lymph nodules show usually nothing but cell hyperplasia. The lesions are not uniformly distributed, but occur in patches throughout the intestine. When present in the stomach, they are of the same kind as those described in the intestine, although rarely so severe. In milder cases the gross appearances may show very little change to the naked eye, except swelling of the lymph nodules. Under the microscope there may be found more or less extensive cell infiltration of the mucosa, but rarely any destructive changes or new connective tissue.

*Ulcerative Form.*—This is rather rare, for the reason that in infancy a very large proportion of the cases die during the acute stage.

The ulcers are nearly always of the follicular variety; occasionally they are broad and shallow. If the patient dies after an illness of from six to eight weeks, the appearances do not differ essentially from those described in acute cases. If life is prolonged from two to four months, ulcers are found in various stages of repair. Follicular ulcers require from one to three months for cicatrisation, and the broad superficial ulcers even a longer time. It is very doubtful whether stricture ever results from these ulcers in children. The mucous membrane shows almost invariably evidences of more or less extensive chronic catarrhal inflammation. Among the very rare lesions are cysts of the colon. Fully developed cysts I have seen but once. The child had an attack of acute ileo-colitis, which became chronic, lasting about five months. He never regained his health, and died one year later from intercurrent disease. In the descending colon and rectum, about twenty cysts the size of a pea, and many smaller ones, were found. They had a thin, translucent covering. On section, a thick, transparent, gelatinous material escaped. They were situated in the submucosa, and were undoubtedly produced by the dilatation of some of the tubular glands whose orifices had been obliterated.

*Associated Lesions.*—The important ones are in the lungs, the most common being hypostatic congestion, subacute or chronic broncho-pneumonia, more rarely pulmonary tuberculosis. It is rare to find the lungs perfectly healthy. The liver is often found extremely fatty in cases associated with great wasting, but in no case have I seen hepatic abscess. The kidneys usually show a more or less intense cloudy swelling, and

sometimes there may be well marked nephritis. Dropsical effusions into the serous cavities are rare.

**Symptoms.**—In the milder cases there are only the symptoms of chronic intestinal indigestion with the constant presence of mucus in the stools, usually in large amount.

The severe cases are usually seen in autumn, and are generally the sequel of acute attacks occurring during the summer.

The signs of active inflammation have passed away; the temperature is usually normal; there is no pain or tenderness. There is, however, no improvement in the general condition, and either the weight remains stationary, or the child continues to lose slowly until it is little more than a skeleton. The face is pinched, the eyes sunken, and the cheeks hollow. The lips are pale, often fissured, and bleed readily. The fontanel is depressed. The body is so small that the head seems much too large. The skin hangs in loose folds on the thighs. The mouth is often the seat of thrush, of catarrhal, herpetic, or rarely of ulcerative stomatitis. The tongue may be heavily coated, but is more often dry, glazed, and red.

Although they seldom cry for food, as a rule these children will take nearly everything given them, and in almost unlimited amount. Notwithstanding that it is retained, the more they are fed the more rapid seems the wasting. Vomiting is not common, and seldom occurs except from overloading the stomach or during acute exacerbations.

The stools are rarely frequent, five or six a day being the average; often there may be only two or three a day for a week at a time. They are thinner than normal, but are not often fluid. They usually contain mucus of a green or brownish colour, often in large quantity, but rarely blood. The stools may consist almost entirely of a green or greenish-brown fluid. They are large in proportion to the amount of food taken. Undigested food is always present in quantity, and upon the diet depends very much the gross appearance of the stool, the odour of which is almost always offensive. Pus is often found under the microscope, but is rarely visible to the naked eye. A form of stool believed to be characteristic of wide-spread inflammation of the mucous membrane with atrophy of the tubular glands is one of nearly normal consistence, homogeneous, dark brown in colour, and very offensive.

Prolapsus ani is not so frequent as in the acute cases; but when it occurs it is generally more difficult to control. Flatulence and colic are prominent symptoms in some cases, but absent altogether in many others. As a rule, there is neither abdominal pain nor tenderness. The abdomen is usually distended, and in most cases the enlargement is uniform, but sometimes there is marked epigastric prominence, which is more often from dilatation of the transverse colon than of the stomach. Although the mesenteric glands are enlarged, they can not be felt through the

abdominal walls. The skin is dry and scaly, and in the worst cases frequently covered with small petechiæ over the abdomen and lower extremities. About the anus, and over the sacrum, thighs, genitals, and sometimes the feet, there are excoriations, and not infrequently ulcerations. The temperature is elevated only during exacerbations, or from inflammatory complications. A subnormal temperature is frequently met with. I have occasionally seen it 95° F. in the rectum. The urine often contains an excessive amount of indican. Dropsy is often present without albuminuria. The weight is stationary, or steadily falls to an almost incredible degree. I have seen one infant weighing but eight pounds at thirteen months; another, thirteen pounds at two years and four months. Ulcers of the cornea are not uncommon. Nervous symptoms are always present. The children are cross and irritable, sleep badly, and frequently have a low, whining cry, which is continued much of the time. Sometimes they are dull, apathetic, and quite indifferent to their surroundings. Persistent opisthotonus is occasionally seen; and there may be contractions of the extremities, but rarely general convulsions.

The duration of the disease is from two months to a year. The progress is irregular, and marked by periods of improvement, during which for a time the patient may hold his own, or even gain in weight. Any trivial cause may excite a relapse, and the downward progress is rapid. Death often occurs during one of these exacerbations, or it may be due to broncho-pneumonia, tuberculosis, or slow asthenia.

**Diagnosis.**—It is important to distinguish the cases with marked cachexia and slow convalescence, although ultimately resulting in complete recovery, from those which present at a certain stage almost identical symptoms, and yet go on steadily downward, terminating fatally. The difference in these cases is really a difference in the character and extent of the lesions. The first group are probably cases of superficial catarrhal inflammation, or of follicular inflammation which has not gone on to ulceration, these lesions being capable of repair. The second group are the cases of ulceration, in which complete recovery from the lesions is impossible, and repair only partial, if indeed any occurs. In distinguishing between these groups the most important guide is the nature of the symptoms during the antecedent acute attack. The longer the acute symptoms have lasted and the higher the temperature, the greater probably is the extent of the lesions, and the more severe their character.

The diagnosis of chronic ileo-colitis from general tuberculosis is often difficult. Except for those whose general condition is extremely bad the differential diagnosis can usually be made by the cutaneous tuberculin test. Tuberculosis is more likely to be met with in institutions, among the poor of cities, and in children previously delicate and with a tuberculous family history.



**Prognosis.**—The prognosis depends upon the child's previous condition, upon the duration of the intestinal symptoms, upon our ability to carry out proper treatment, upon the presence of complications; but, most of all, upon the severity and extent of the intestinal lesions. The possibility of error always exists in estimating the gravity of the lesions, so that no case should be considered hopeless. The most unpromising cases sometimes end in complete recovery. If, however, continuous symptoms have existed for eight or ten weeks without any sign of improvement, recovery is extremely doubtful. The patient may linger for two or three months longer, but usually only to be carried off by the first acute disturbance which occurs.

**Treatment.**—No greater mistake is made than to give these children week after week the various diarrhoea-mixtures, with the expectation that ultimately the formula which exactly meets the particular case will be found. Drugs are to be used only for the relief of special symptoms. Thus a dose of opium may be needed when the movements are unusually frequent, or castor oil, or calomel occasionally when the stools are particularly offensive. The essential and important part of the treatment consists in injections, careful feeding, and change of air. Astringent enemata, however, are of some value. They should not be given continuously, but from time to time should be omitted for a week or two to see what the condition of the stools is without them. I have seen several cases of the milder variety where the constant use of such injections seemed to be an important factor in keeping up the production of mucus. The colon should first be washed with a large amount of a tepid salt solution, and then four or five ounces of the astringent solution injected, and held in place by compressing the buttocks for half an hour.

Alcohol is often useful but it should be given in moderate amounts and well diluted. Port or sherry is often better than brandy or whiskey. The diet advised for later stages of the acute cases should be continued. Fat and starchy foods should be excluded for a long time and then given in small quantities and cautiously. The fat of cow's milk especially should be avoided; olive oil, however, can usually be given at an earlier period and in many cases is borne surprisingly well. Kumyss and zoolak, skimmed milk, and buttermilk are useful. To these articles may be added, beef juice, rare scraped beef, and the whites of fresh eggs, partially cooked. The diet should be directed according to its effect upon the stools. Much information may be obtained by thoroughly washing the stools and examining the residue. Nutrition may be promoted to some degree by inunctions of cocoa butter, cod-liver oil, or some other form of fat.

The patients should be placed in the best possible surroundings; in no disease is a change of air more to be desired than in this. They should be in the open air as much as possible but should be kept warm,

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for their temperatures quickly fall to subnormal. The danger of relapses and acute exacerbations continues long after the primary attack has subsided.

#### AMŒBIC COLITIS.

Amœbic colitis is rare in children; it is particularly so in infants, probably owing to the fact that nearly all the water taken at this age is boiled. Most of the cases in children thus far reported have been observed in warm climates, although Amberg has recorded five which occurred in Baltimore, the youngest child being two years and eight months old.

The symptoms in the few cases that have been reported in children have differed in no important particular from the disease as seen in adults. In exceptional cases the onset may be abrupt and the attack may run an acute course, terminating fatally in two to three weeks. Such cases are characterised by much abdominal pain and tenderness, frequent mucous and bloody stools containing amœbæ, and some fever, which, however, seldom reaches 102° F.

More frequently this acute onset is followed by a subacute or chronic form of the disease, or the disease may be subacute from the beginning. The protracted cases are the type of the disease most frequently seen. They are very obstinate to treatment. Periods of constipation and apparent recovery often alternate with exacerbations in which the bloody and mucous stools return, with pain, tenesmus, and slight fever. The duration may be from a few months to one or two years. Death may finally occur from exhaustion with extreme wasting, or from some complication, such as hæmorrhage, abscesses of the liver being very rare in children. The diagnosis from other forms of colitis is made only by the discovery of amœbæ in a freshly voided stool.

The general treatment is the same as for other forms of acute or subacute colitis. The special treatment for the purpose of destroying the amœbæ is the use of injections of quinine which may be employed in solutions varying in strength from 1 to 5,000 to 1 to 250.

#### AMYLOID DEGENERATION OF THE INTESTINES.

This is rarely met with in infants. It is not so infrequent in older children, where it is associated with amyloid changes in the liver, spleen, and kidneys, usually as a result of prolonged suppuration in connection with bone tuberculosis. It is sometimes met with in syphilis. The ileum is the part of the intestine most affected. The process begins in the walls of the arterioles and capillaries, particularly of the villi, and later involves the vessels of the submucosa; subsequently the epithelium may be affected. The mucous membrane in these cases is pale, somewhat

translucent. The reaction is reversed by the application of the iodine test; the affected area becomes of a brownish-red or mahogany colour.

Any local degenerative processes or definite symptoms. Flatulence is frequent but is of no means constant. The abnormal and wary bacteria which are present are probably dependent more upon the associated lesions of the liver and kidneys than upon the changes in the intestines.

#### TUBERCULOSIS OF THE INTESTINES AND MESENTERIC LYMPH NODES, MESENTERIC GLANDS

These two conditions are usually, but not invariably, associated and may be conveniently considered together.

**Frequency.**—In a series of 606 autopsies upon tuberculous cases from my own hospital records the intestines were involved in 40 per cent. The great majority of the patients were under three years of age. In 141 autopsies upon tuberculous cases published in the *Pediatrics Hospital Reports*, the intestines were involved in 5 per cent. These patients were mainly between four and 6 years of age. In 89 autopsies upon tuberculous children, chiefly infants, reported by Miller, the intestines were involved in 75 per cent. In 143 autopsies collected by Becker there were intestinal lesions in 31.6 per cent. These figures show that tuberculosis of the intestines is not one of the most frequent forms in children, and that it is rather less frequent in infancy than at a later age. It is most common from the third to the eighth year. The mesenteric lymph nodes were tuberculous in 44 per cent of my own autopsies, and in 35 per cent of the *Pediatrics* cases, showing thus in both series with slightly greater frequency than tuberculosis of the intestines.

**Etiology.**—In the great majority of cases the mesenteric lymph nodes are infected from the intestines. It is possible, but I believe exceptional, for the infection to come through the general circulation. When tuberculosis exists in the intestine the lymph nodes are constantly found by inoculation in animals to be tuberculous, although they may not yet be caseous. The infection of the mesenteric lymph nodes sometimes is from bacilli in the milk. Much stress has been laid upon tuberculosis milk as a means by which children are infected. Primary tuberculosis of the intestine is in this country relatively a rare condition. I have records of less than a dozen such cases in nearly four hundred autopsies upon tuberculous patients. When it does occur, however, primary tuberculosis of the intestine has been in my cases due to the use of a bacillus of the bovine type. If the human type the infectious condition is probably produced from tuberculosis milk was the source of the infection. The intestinal lesions most frequently found are, however, of a chronic and non-inflamed type and probably secondary to the inflamed pal-

monary process. They are doubtless due to swallowing tuberculous sputum. In such cases the human type of bacillus is found.

**Lesions.—Intestines.**—The usual seat is the small intestine, chiefly the jejunum and lower ileum. With extensive disease the large intestine may also be involved, most frequently the cæcum, and exceptionally it alone may be affected. Tuberculous ulcers may be found in the appendix.

The early deposits appear as tiny yellow nodules, generally widely scattered and affecting Peyer's patches. Usually, however, ulcers are present, and often only ulcers are seen. Their size and number vary greatly; there may be only five or six tiny ulcers, or there may be forty or fifty, the largest being two or three inches in diameter. They very frequently involve Peyer's patches. The typical tuberculous ulcer is of irregular shape, with rounded borders and with its longest diameter at right angles to the intestinal axis. When large, it may nearly encircle the gut. The ulcers are excavated; they have overhanging, infiltrated edges of a deep-red colour. The surface is covered with granulations. In those which have partially healed a distinct puckering of the intestine occurs, which is especially noticeable upon the peritoneal surface. The small ulcers involve the mucosa only; the larger and older ones the submucosa and the muscular coats, and not infrequently also the serous coat. Perforation may occur, but rarely into the general peritoneal cavity, as a localised plastic inflammation precedes it. There may be adhesions of adjacent intestinal coils, and fistulæ may form, owing to ulceration at the point of contact. With these severe cases there is always associated more or less extensive tuberculous peritonitis, frequently of the ulcerative variety. Like other tuberculous processes, the infiltration and ulceration may cease at any stage, and cicatrisation follow. If the ulcers have been large ones, there is always some narrowing of the lumen of the intestine. Stricture is rarely seen because most patients die from the general disease before it has had time to occur. Monti has reported a case of obstruction at the ileo-cæcal valve, due to an old tuberculous cicatrix, in an infant of twenty-one months. One has come under my observation in a child of nine years, in which the obstruction was in the colon, just beyond the ileo-cæcal valve.

**Mesenteric Lymph Nodes.**—Usually these tuberculous lymph nodes are from half an inch to an inch in diameter; occasionally they may reach the size of a hen's egg. From a fusion of several of them, tumours of considerable size may be formed. I have seen one such mass as large as the head of a child at birth.

The process is the same as that which occurs in other lymph nodes of the body. There is a tuberculous inflammation, followed by caseation, softening and abscess, or by calcification. Localised peritonitis is found in all the marked cases; this is usually plastic, but may be suppurative

when due to the rupture of an abscess. Pressure upon the *vena cava* may lead to dropsy in the lower extremities. Ollivier has reported a case in which thrombosis of the *vena cava* occurred. Pressure upon the portal vein may lead to ascites and dilatation of the superficial abdominal veins. There may be pressure upon the thoracic duct.

**Symptoms.**—The symptoms of intestinal tuberculosis are exceedingly irregular. Ulcers are very frequently found at autopsy when there have been no marked intestinal symptoms; this is especially true of the small ulcers usually seen in infants. On the other hand, diarrhœa is not uncommon in cases of advanced general tuberculosis where no ulcers are present. It is the most frequent symptom, and may be exceedingly obstinate. The stools do not differ essentially from those in chronic ileocolitis, except in the occurrence of hæmorrhages and in the presence of tubercle bacilli. Hæmorrhages are not very frequent, but they may be so large as to be the cause of death. This occurred in one of my cases, an infant nine months old, the blood coming from a single ulcer in the ileum. Hæmorrhage is more common in older children. In some cases localised abdominal pain or tenderness is present. In advanced cases the symptoms of intestinal ulceration are usually mingled with those of peritonitis, and there are also present the enlarged mesenteric lymph nodes, which may aid in the diagnosis. In the vast majority of cases, these nodes are recognised only by deep palpation. The tumours are generally felt as irregular nodular masses, lying close against the spine, not movable, and sometimes tender on pressure. Other tumours from deposits in the peritoneum may be present anywhere in the abdomen; they may be superficial or deep. The other symptoms are due to the complications already mentioned and to tuberculosis elsewhere.

**Diagnosis.**—The only positive evidence of intestinal tuberculosis is the discovery of the bacilli in the stools. They are here to be carefully differentiated from smegma and other forms of acid-fast bacilli. In the absence of such evidence, the disease is differentiated from simple ileocolitis, first, by the signs of tuberculosis elsewhere in the body, especially in the lungs, these being almost invariably involved; secondly, by the slow onset and gradual development of the symptoms, while in chronic ileocolitis an acute attack has almost invariably preceded. Large hæmorrhages always suggest tuberculosis. A positive reaction to the tuberculin test is of much assistance in diagnosis.

The large mesenteric glands are recognised only as abdominal tumours.

**Prognosis.**—This depends altogether upon the extent of the tuberculous disease elsewhere, as it is extremely rare for the intestinal lesion to be the cause of death. Once formed, the ulcers probably remain, cicatrization being very rare, and then only partial.

**Treatment.**—The only symptom which ordinarily demands treatment is the diarrhoea. When severe, this is to be managed much as in cases of ileo-colitis, except that irrigation of the colon is, of course, not called for. The chief reliance must be upon diet and internal medication. The drugs which are most useful are bismuth, opium, and creosote; the last mentioned should be given in pills coated with shellac.

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## CHAPTER VIII.

### *DISEASES OF THE INTESTINES.—(Continued.)*

#### CHRONIC INTESTINAL INDIGESTION.

As the larger and more complex part of the process of digestion goes on in the intestine, intestinal indigestion is a more common and more complicated disturbance than is gastric indigestion. In many cases we find the two associated, but in perhaps the majority the symptoms relate entirely to the intestinal process. The conditions seen in young infants are so different from those in older children that the cases may be best considered separately.

#### *In Young Infants.*

The general causes are the same as those mentioned in connection with chronic gastric indigestion: constitutional debility, either congenital or acquired, unfavourable surroundings, and previous attacks of acute disease. Chronic intestinal indigestion is especially common during the first six months, and is seen both in nursing infants and in those who are artificially fed. In the case of breast-fed infants, the mother is often highly nervous, delicate, and anæmic, and may be taking large quantities of fluids of every description, for the purpose of maintaining an abundant flow of milk. Why it is that the milk causes so much disturbance can not always be discovered even by the most careful analysis. Sometimes the trouble is simply that the milk is too rich, chiefly in fat. Disturbances may come, although rarely, from over-feeding.

In infants who are being fed upon cow's milk, the most common cause is that the fat is excessive; sometimes it is the sugar, and it may be both. When once begun a striking intolerance of both fat and sugar persists for a long time. Another very important cause is the use of farinaceous foods too early, in too large quantities, and often insufficiently cooked.

**Lesions.**—Strictly speaking, chronic indigestion is a functional disorder without anatomical changes. When the condition has lasted for

many weeks or months, as often happens, there may result a low grade of catarrhal inflammation in the colon, frequently attended by hyperplasia of the lymph nodules of the mucous membrane, and sometimes by a similar process in the mesenteric lymph nodes. Chronic indigestion may be the principal and the only symptom in cases of chronic ileocolitis which follow acute attacks.

**Symptoms.**—The general symptoms are those of malnutrition, or in the more severe form, those of marasmus. These have already been fully described, and need only be mentioned here. The most important are, stationary or falling weight, anæmia, poor circulation, often subnormal temperature, almost constant fretfulness and crying, with very little quiet sleep. The tongue may be coated or quite clean. The appetite is often good, these infants taking food whenever given, and in an almost unlimited quantity. There are few cases in which occasional vomiting does not occur, but it is rarely persistent.

So far as the intestinal condition is concerned, the cases may be divided into those with diarrhœa and those with constipation. It may happen that the same child will suffer for a long time from diarrhœa and then from constipation, or the reverse; but usually one condition or the other is habitual. The diarrhœal stools are thin, green, and contain undigested food and mucus. They vary in number from three to six or eight in twenty-four hours. They are commonly passed without pain, although there may be flatulence. The stools have usually a sour, unpleasant odour, but they are rarely foul. They may be irritating to the skin, and cause troublesome excoriations or intertrigo. In some cases the stools contain but little solid matter, the character being that of yellowish-green water. In most of the cases, after the process has lasted two or three weeks, mucus is present, and may then become a constant feature.

If there is constipation, the stools are usually gray or white; they are smooth and pasty or like hard balls and passed after much straining, often coated with mucus and sometimes streaked with blood. These stools contain an excessive amount of fat, especially in the form of soaps and also a larger proportion of inorganic matter than is normal, particularly calcium salts. Often the bowels will not move for days except after the use of laxatives or enemata. The latter frequently have but little effect, as the rectum may be empty. Constipated cases are especially prone to suffer much from flatulence and colic, the attacks of which may be very severe.

The duration of these symptoms is indefinite. There is little or no tendency to spontaneous improvement, and they may drag on for several months or until the problem of diet is solved. The progress of these cases is marked by frequent exacerbations, during which there is vomiting, and usually fever. Such symptoms are generally dependent upon

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intestinal toxæmia. A low irregular fever may continue for days or even weeks. Although the general symptoms of failing nutrition are present in most cases, a mild degree of chronic intestinal indigestion with frequent loose movements may sometimes last for months, during which the patients may gain steadily in weight and give every indication of being well nourished. This is much more common in nursing infants than in those who are artificially fed.

**Diagnosis.**—It is not generally difficult to determine that an infant is suffering from chronic intestinal indigestion; but one should endeavour to go further in his diagnosis and discover which of the elements of the food is causing the chief disturbance. Much valuable information may be gained from a careful history of what has already been tried in the case; often some gross error can be detected in the proportions of the food elements, the quantity of food given or its preparation. Difficulty with the fat is sometimes indicated by loose movements, usually of a yellow or yellowish-green colour. Sometimes they are clay coloured, smooth and formed, with a peculiarly offensive odour; there may be vomiting or the regurgitation of food in small quantities. Difficulty with the sugar is often associated with flatulence, colic, and diarrhœa, with thin, sour, irritating stools. Difficulty with the starch leads to much flatulence and colic, diarrhœa alternating with constipation, and offensive stools. One may find the foregoing symptoms in any combination, for although in the beginning the trouble may be with but a single element of the food, this is rarely true when the child comes under observation. By carefully noting the symptoms which follow the use for a few days of a simple milk formula, such as fat 1 per cent, sugar 5 per cent, protein 0.9 per cent (one-fourth whole milk), one can often arrive at a conclusion as to which element of the food is producing the most disturbance.

**Prognosis.**—This depends almost entirely upon how early the cases come under treatment and how they are managed. There is very little tendency to spontaneous improvement or recovery. The outlook is much better in cases with constipation than in those with diarrhœa. In the latter, progress is very difficult as the intolerance of food is so great that increase in weight is well-nigh impossible. The existence of chronic intestinal indigestion is one of the most important predisposing causes of more serious forms of intestinal disease.

**Treatment.**—Drugs have no part in the treatment of these cases, except now and then for particular symptoms, such as diarrhœa, constipation, or colic. These infants are cured by proper dietetic and hygienic measures, and by these alone. The diet has already been discussed in the chapter on Infant-Feeding, and the general management, not less important, in the chapter on Malnutrition.



*In Older Children.*

Chronic intestinal indigestion is especially common in children from the first to the fifth year. With the younger children, solid food has generally been given too early and in too large quantities. The articles from which most trouble is seen are imperfectly cooked cereals, vegetables of all kinds, but especially potato. Often the diet is composed almost entirely of farinaceous foods and bread. The condition often follows an attack of acute diarrhoea or dysentery. Children suffering from rickets are particularly liable to be affected. The disease is seen in all grades of society.

**Symptoms.**—The clinical picture which these cases present is a very common one, and the symptoms are quite uniform. The patients are generally very thin with very small extremities, a small amount of fat,



FIG. 64.—CHRONIC INTESTINAL INDIGESTION. — Patient four years old; symptoms of three years duration, following attack of acute ileo-colitis. Height, 34 inches; circumference of abdomen, 24 inches; weight 24 pounds.

and a large prothorax and chest (Fig. 64). There is much flatulence, and usually there is marked tympanites. Such children are pale, anorectic, and sallow in complexion; they have dark rings under the eyes; they are fatigued on slight exertion; they are very cross, irritable, and emotional to an unusual degree. They are hard to amuse, hard to control, and altogether exceedingly difficult patients to deal with. Their growth is retarded if the symptoms have lasted long. They are much below the average in height and weight, but mentally often quite precocious. One of my patients at five years weighed twenty-two pounds and was thirty-three inches tall. The sleep is always unnatural and disturbed; and at night they toss about their beds, waking frequently, crying out and often grinding their teeth, this sometimes leading to the diagnosis of intestinal worms. They perspire very readily, and suffer from cold extremities.

The bowels are usually constipated, the stools being of a light gray colour or nearly white. The odour from the discharges is

usually extremely foul. This condition may alternate with diarrhoea. The stools are then not very frequent, rarely exceeding four or five a day, but they are large, gray, green, or brown in colour, often foetid, offensive, and always contain undigested food. They are in many cases excited by the

taking of food. From time to time, in many patients, large quantities of mucus are passed; in some cases this comes to be a constant feature of the disease. Large quantities of gas are expelled. Pain is not a very common symptom in most cases. The appetite is capricious and usually poor, though some patients will eat everything offered. The tongue may be coated; but unless the stomach is also affected it is usually clean and the breath is not offensive.

The nervous symptoms which these patients present are exceedingly varied, and often of the most puzzling character. In many cases they are so severe and so persistent as to lead to the diagnosis of organic disease of the brain. In addition to the condition of general nervous irritability, there may be tetany, fainting attacks resembling somewhat the seizures of *petit mal*, exaggerated reflexes, attacks of dulness or sometimes stupor, with retracted abdomen, irregular pulse and respiration, and other symptoms strongly suggestive of tuberculous meningitis. Convulsions are not uncommon. They are usually accompanied by fever, and may be repeated at intervals of a few minutes. There is almost no end to the combinations of nervous symptoms which these patients may present. Most of them are toxic in their origin. The skin shows frequently eruptions of erythema or of urticaria.

Slight fever is sometimes present for weeks, the temperature usually varying between 99° and 101.5° F. Sometimes for several days it may be normal, and occasionally may rise to 102° or 103° F. during a slight exacerbation in the symptoms. The urine of most of these patients contains a great excess of indican; the amount present indicates very accurately the degree of intestinal putrefaction, and often fluctuates regularly with the nervous symptoms.

Intercurrent attacks of acute indigestion, with diarrhoea and vomiting, are common and quite easily excited. The course and duration of these symptoms are indefinite. In the most severe forms, if untreated, the patients gradually waste until they die from exhaustion, or fall easy victims to any acute disease which they may happen to contract. There is but little tendency to spontaneous recovery.

Herter has called special attention to a type of this disease associated with marked arrest in growth to which he gave the name Intestinal Infantilism. In several such cases studied he found a failure of retention of calcium and magnesium salts over a prolonged period of time. To this he ascribed the arrested development of the skeleton. Associated with this, there were present in all cases evidences of excessive intestinal putrefaction. The bacteriology of the condition he believed to be characteristic, viz., a preponderance of the *b. bifidus*, with great diminution or entire absence of the *b. coli*.

**Prognosis.**—This depends upon the duration of the symptoms, the general condition of the patient at the time treatment is begun, and upon

how thoroughly it can be carried out. The symptoms, in the great majority of cases, have existed for several months at the time the case comes under observation. Generally, the greater the mistakes in feeding have been, and the greater the violation of hygienic and dietetic rules, the better the prognosis. A child who has developed chronic intestinal indigestion of a severe type, in spite of the fact that the hygienic surroundings were good, and when the dietetic errors were not flagrant, is not nearly so hopeful a subject for treatment as one whose hygienic surroundings have been poor and whose diet has been especially bad. In cases like the latter, a removal of the causes and the institution of proper methods of treatment almost invariably result in immediate and striking improvement, unless the general vitality of the patient has been reduced to a very low point. In the other cases, where the mistakes have been less marked, and the condition is due more to constitutional than to local causes, the improvement is slower and less striking. Thus, as a rule, hospital patients improve more rapidly than those seen in private practice.

**Treatment.**—In no class of cases that the physician is called upon to treat are results more satisfactory than in many of those of chronic intestinal indigestion, when intelligent co-operation can be secured. If the parents themselves are lax in discipline, and are unable to control the child, an efficient trained nurse should be secured, into whose hands the exclusive management of the child should be placed. The essential part of the treatment is diet and general management. In the second and third years the most important thing is to stop all starchy food for a considerable time, and put the patient upon an exclusive diet of rare beef or beef juice and skimmed milk or buttermilk. After some improvement has occurred carbohydrates may be added, some of these in the form of maltose, but chiefly as a well-cooked starchy food. The number of feedings should not be more than four a day during the second year, and three or four a day for children during the third and fourth years. These should always be at regular intervals, and nothing whatever given between meals. The meat should be rare scraped beef-steak or mutton chop; from one to three tablespoonfuls may be allowed once a day. The white of egg may be given early, and after a time, the whole egg. Kumyss and zoolak and buttermilk are often of very great value. Although at first they are taken with difficulty, in many cases a fondness for them is very soon acquired.

After improvement has been going on for two months, bread may be added, at first in small quantities and once a day. This should preferably be stale, cut thin and dried in the oven until it is crisp, and given without butter. Two or three times a week raw oysters may be tried. Mutton, chicken, or beef broth, without vegetables, may be given occasionally in the place of one of the milk feedings. After this diet has

been kept up for three or four months, if improvement continues, one of the green vegetables thoroughly cooked and strained may be added once a day. A striking feature of these cases is their marked intolerance of the fat of cow's milk. This must be withheld for a long period. The form of fat which these patients can take best is usually olive oil, which furnishes a valuable means of increasing weight. Beginning with one teaspoonful three times a day the quantity may be increased to two or three times this amount. This restricted diet should be continued for at least a year or until all the symptoms have disappeared. Potato and catmeal should be forbidden for a long time.

Intestinal irrigation is useful for brief periods in some cases in which there is much mucus passed. But it should not be forgotten that continued irrigation often keeps up the production of mucus. Astringents should not be used, but only a warm saline solution.

The constipation can sometimes be controlled by the diet alone; but in most cases drugs are needed also. Calomel frequently seems to exert a very beneficial influence, even when the constipation is not severe. It is often wise to administer a full dose every week or ten days. In some patients castor oil acts more satisfactorily. It may be objectionable, however, from its tendency to aggravate the constipation. As laxatives in this condition I have found the greatest satisfaction from the use of preparations of cascara and the compound licorice powder. Abdominal massage is also useful.

Drugs directed against the process of putrefaction are extremely unsatisfactory even in older children, but sometimes diminution in the amount of flatulence follows the use of subgallate of bismuth, carbonate of creosote, salol, or salicylate of soda. General tonics are required, and may add materially to the improvement of the patients. Altogether the best is *nux vomica*. It may be given in combination with the bitter wine of iron just before meals three times a day. Cod-liver oil, particularly in the early stage, is badly borne.

Relapses are easily excited by indiscretion in diet, and parents should be impressed at the very beginning with the necessity of adhering rigidly to the diet prescribed, for a long period. It very often happens that the improvement which is seen after one or two months of careful treatment is so marked as to lead the parents to the belief that a cure has been accomplished, so that they relax their vigilance and allow improper articles of food which are almost certain to induce a relapse. If the case is an aggravated one, and the symptoms of long standing, it is wise to tell parents at the outset that a year's treatment is the minimum in which anything permanent can be accomplished.

The general treatment of the patient must not be overlooked. Proper clothing, regular exercise in the open air, cool sleeping rooms, massage, and sponging every morning with cold water, are all of very great im-

portance, and contribute almost as much to the results obtained as the special measures adopted. (See chapter on Malnutrition.)

An elastic abdominal bandage giving moderate support not only adds to the comfort of these patients but to some degree prevents the excessive distention likely to occur on account of the loss of muscular tone in the abdominal walls.

The improvement in the nervous symptoms of the patient is one of the first things noticed, and is often marked in a few days after the beginning of treatment. From an irritable, fretful, peevish child the patient is sometimes totally changed in disposition in a few weeks, so as to become quiet, affectionate, docile, and playful.

#### INTESTINAL COLIC.

The term *colic* is applied to any severe paroxysmal pain occurring in the intestines. It may be due to many causes. The colic of lead and arsenic poisoning are both very rare in children; but colicky pains are present in appendicitis, intussusception, ileo-colitis, and, in fact, in all the severe forms of intestinal inflammation. Colic may be due to swallowing certain substances, especially foreign bodies and the seeds of fruits; and in rare cases it may be excited by the presence of round-worms when they are numerous. In all the conditions mentioned, colic is only one of the symptoms, although it may be a very prominent one.

The peculiar colic of infancy is clearly caused by spasm of the muscular wall of the intestine. It is a heightened reflex from irritation of which we have many other illustrations at this period of life. The cause of the irritation is usually the presence of some undigested food in the intestine. Colic is therefore essentially a symptom of indigestion. Flatulence and colic are very often, but not always, associated. Colic is always increased by the coexistence of constipation, which in many cases is its sole cause. Almost any of the elements of the food may give rise to colic.

Sugars and starches may produce it by causing excessive fermentation and flatulence. Fats are less frequently at fault; but the presence of large unabsorbed masses in the intestine may be a sufficient cause of irritation. The actual pain in colic is partly from distention, but chiefly from muscular spasm. In some of the most severe cases of colic it is possible that the spasm may be accompanied by a slight transient intussusception. Colic may follow chilling the surface of the body. In these cases, also, muscular spasm appears to be the principal factor in causing the pain. The colicky period of infancy is chiefly the first three months; after this time the peculiar susceptibility gradually passes off.

**Symptoms.**—These are in most cases so typical as to be easily recognised. They are always more severe in delicate and highly nervous children. In the severe attacks there is contraction of the features, a loud paroxysmal cry, subsiding for a few moments and then beginning with renewed intensity, drawing up of the lower extremities, and in male infants contraction of the scrotum. With these symptoms the abdomen is usually found tense and hard. With the expulsion of the gas, the symptoms subside at once, and the child usually falls asleep. In the most severe attacks there may be considerable prostration, cold extremities, and perspiration. When the symptoms are less severe there is only continual fretfulness, and the child can not sleep. When colic is habitual there are very few hours in the twenty-four when the child seems to be entirely comfortable. In nursing infants there may at times be difficulty in distinguishing the cry of colic from that of hunger, as infants suffering from colic will usually take food eagerly, and this is often followed by temporary relief. In colic, however, the pain soon returns, and often is more severe than before. The cry of colic is usually violent and paroxysmal; that of hunger is apt to be prolonged and continuous, and is not accompanied by the other symptoms mentioned as indicating abdominal pain. In older children the less frequent causes of colic mentioned at the beginning of this article, especially appendicitis, should be borne in mind.

**Treatment.**—When colic is due to flatulence of the intestine, nothing given by the mouth has much effect in relieving the symptoms. Certainly food should not be given. The purpose of treatment during the attack is to assist the child to get rid of the gas; as this is usually in the colon, the most efficient means is by massage or enemata. At first an injection of four or five ounces of lukewarm water should be used. If this is not successful, two ounces of cold water with half a teaspoonful of glycerin may be tried. This rarely fails to start peristalsis and expel the gas. In conjunction with these measures, dry heat should be applied to the abdomen by means of hot flannels or a hot-water bag, and the feet should be well warmed. In cases of colic not associated with flatulence, when the pain is probably the result of muscular spasm, opium in some form is required in addition to heat or counter-irritation. The treatment between the attacks and the treatment of habitual colic should be directed toward the constipation and the indigestion, upon which they depend.

#### CHRONIC CONSTIPATION.

Constipation may be said to exist whenever the stools are less frequent, harder, and drier than normal. During the first six months infants usually have two movements a day. Many, however, have only one; but if this is normal in character the child is not constipated. In other

cases, although there are two and even three stools a day, they may all be small, dry, and hard, having all the characters of constipated stools, and the case should be treated accordingly.

**Etiology.**—The causes of chronic constipation are many and far-reaching. It may be due to a diminution in the secretion of the intestinal glands or of the liver. The movements are then hard, dry, very light-coloured, and are associated with much flatulence and other signs of intestinal indigestion. Very often the principal factor in constipation is insufficient muscular contraction in the intestine. The faecal masses are then propelled so slowly and remain so long in the intestine that the fluid portion is absorbed, the residue becoming, in consequence, so dry and hard that it is difficult to expel. In other cases constipation is due to the fact that there is insufficient volume to the stools, as may be the case when the food leaves very little residue. Constipation may depend also upon local causes, as, for example, where an evacuation of the bowels is resisted on account of pain from fissure of the anus or from hæmorrhoids. Although not the primary cause, this condition may be sufficient to keep up the constipation indefinitely. It may, in rare cases, be due to a congenital condition, such as narrowing or twisting of the large intestine at some point. Another rare cause seen especially in infancy is tonic spasm of the anal sphincter. The most important causes of constipation may be grouped under two heads: diet, and conditions giving rise to muscular atony.

**Diet.**—In breast-fed infants the trouble is usually a lack of fat and low total solids in the milk. In those who are artificially fed it is often because the fat is too low, and sometimes because both the fat and the protein are too low, the stool lacking volume. In other cases the cause of constipation is indigestion, in still others the use of sterilised milk. During the second and third years the cause may be too much cow's milk, particularly that which has been boiled, or the use of an excessive amount of starchy food. In older children the cause may be an excess of starchy food and a lack of sufficient green vegetables, meat, and fruit.

**Muscular Atony.**—The most common cause of muscular atony is habit; in a large number of cases lack of proper training is the principal etiological factor. If the inclination to have a stool is regularly disregarded it soon ceases to be felt. The ordinary irritation from faecal masses produces no response whatever. The longer such a condition continues the more obstinate does it become. This is an important factor in all cases. Another potent cause of muscular atony is rickets. In this disease the muscular walls of the intestine suffer like the muscles of the extremities, and become incapable of doing their work. Again, any form of malnutrition in which there is feeble muscular tone may cause or aggravate constipation. It is often seen as a sequel to acute

attacks of diarrhoeal diseases, particularly when these have been prolonged. Want of sufficient muscular exercise is a frequent cause. There are many children who rarely suffer from constipation in summer when they have plenty of out-of-door exercise, who very often do so in winter when such exercise is wanting. A loss of muscular tone is not an infrequent result of the prolonged and indiscriminate use of purgative drugs or enemata.

**Symptoms.**—In many cases no symptoms are present except the local ones, the general health being excellent and the nutrition in no way disturbed. In the majority, however, there are symptoms of greater or less severity, depending somewhat upon the cause of the constipation. There may be simply flatulence and colicky pains, or the irritation of the hardened faecal masses may produce a slight catarrhal inflammation of the sigmoid flexure and the rectum, so that mucus and sometimes traces of blood may be passed with the stool. Hæmorrhoids may develop even in infancy, and frequently the constant straining leads to the production of hernia. In many cases there are from time to time nervous symptoms resulting apparently from the absorption of various toxic materials from the intestine. There may be headache, dulness, fretfulness, disturbed sleep, and associated signs of intestinal indigestion. The urine often contains indican in excess, and there may be slight fever.

**Diagnosis.**—This includes the discovery of the cause and the principal seat of the constipation. To arrive at the former the most careful and thorough investigation should be made of the child's diet and habits. It is desirable to determine whether the seat of trouble is the rectum, the upper part of the colon, or the small intestine. If a suppository is almost immediately followed by a normal stool, one may be sure that the rectum only is at fault, and that it needs but a little extra stimulus to make it do its work. This is common in infants who are too young to make any voluntary efforts. In such cases there are no other symptoms present. In others, the white or gray stools, marked flatulence, offensive breath, and general irritability, leave no doubt of the fact that the trouble is due to indigestion.

**Treatment.**—This is always difficult, and in obstinate cases must be continued for a long time. The co-operation of an intelligent mother or nurse is absolutely indispensable. To establish the habit of regular stools should be the first step, for without this regularity nothing can be done. Even in infants only a few months old proper habits are often easily formed if the child is put upon the chamber or chair invariably at the same hour. When a local stimulus is required in addition an oiled glass rod or a gluten suppository may be inserted. An older child must be taught to heed the first impulse to evacuate the bowel. Regular habits can hardly be formed unless the same time each day is chosen for the movement. That to be preferred is soon after the morning meal,



as taking food into the stomach usually starts a peristaltic wave which is continued throughout the intestine. With older children breakfast should be early enough to allow ample time for this duty before the other engagements of the day; and nurses should be impressed with the importance of the early formation of proper habits on the part of their charges. Stretching the sphincter under an anæsthetic is sometimes of great benefit, especially where tonic spasm is present.

*Food.*—With nursing infants who get good breast-milk constipation is rare. When the milk is low in fat, constipation is not uncommon. For the measures by which such milk can be improved, see chapter on Breast Feeding.

In feeding cow's milk, constipation is overcome by getting the proportions of protein and fat which are suited to the infant. It is more apt to occur with infants where, on account of digestive symptoms, modifications of whole milk or skimmed milk are given instead of those from top-milk. The laxative effects of maltose and, to a less degree, of lactose, should be remembered (see Infant Feeding). With most infants during the first year, constipation may be, if not cured, at least prevented, by proper milk modification.

During the second year children who suffer from constipation may be benefited by reducing the amount of milk and giving a limited quantity—not over three or four ounces a day—of thin cream. Improvement may often be brought about by using the coarse farinaceous foods. Meat broth and beef juice are quite laxative on account of their extractives and salts. Fruits are valuable in all these cases; but only the juice should be given until a child is eighteen or twenty months old. That of almost any fresh fruit may be employed. At two years pulpy fruits may be given, but only after cooking; also baked apples, stewed prunes, and, in summer, peaches, plums, and pears, in small quantities; but berries should be avoided. Fresh fruits should not be given until after three years and then in moderate quantities only.

For older children who are on a mixed diet the amount of starchy food should be moderate. Milk should be given rather sparingly. It is sometimes advisable to stop milk altogether and give only cream, from three to four ounces of which may be allowed daily. It may be used with the breakfast cereal, mixed with potato or rice, added to soups or broths, and taken in various other ways. All bread should be made from whole wheat or unbolted flour. Bran biscuits are also useful. Meat and broth may be allowed freely, also green vegetables, one of which should be given every day. All fruits allowed infants may be used, but in larger quantities, and in addition raw apples. Of the dried fruits, only dates, prunes, and figs are permissible, and these only after cooking. Fresh fruit is preferably given in the morning, oranges being especially useful when taken on rising. A caution is necessary in the use of fruits and

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coarse foods for constipated children. It often happens that constipation is only one of the symptoms of a chronic intestinal indigestion, and the use of such foods as those mentioned, while it may cause the bowels to move, aggravates the primary condition. They produce abdominal pain, flatulence, and the discharge of mucus in the stools. The administration of some mild laxative even over a considerable period is often much less objectionable.

Either hot or cold water, when taken an hour before breakfast, may be of considerable benefit to older children. The sparkling waters, like Vichy or Apollinaris, are often better than plain water.

Massage, when properly employed, is useful in conjunction with other measures, but rarely succeeds alone. It should be given for five or ten minutes after retiring and just before rising.

A proper amount of active muscular exercise is necessary and should be made a part of the treatment in every case. Yale has called attention to the importance of posture during the stool, he having found that in many cases a cure was effected simply by substituting a low seat on a nursery chair or closet for the high one previously used.

*Suppositories.*—In many cases, particularly in young infants who are not old enough to initiate the muscular effort, a slight stimulus to the rectum is all that is required. The cone of oiled paper has a great reputation in domestic practice and is not objectionable. It may be of assistance in establishing the habit of a daily movement at a regular time. Soap suppositories produce a more marked irritation; although their immediate effect is quite satisfactory, they should not be continued indefinitely. They are, however, less objectionable than glycerin suppositories. The latter, for an immediate effect, are convenient and usually efficient; but their frequent use, especially in infants, is likely to set up a catarrhal proctitis. The gluten suppositories produce less irritation and are consequently slower in their effect, but they have not the disadvantages of the soap or glycerin. Medicated suppositories are often efficient; if drugs must be employed, they are perhaps open to the fewest objections when used in this way. The following are the best drugs for this purpose, the dose being that for a child of two or three years: ext. nux vomica, gr.  $\frac{1}{12}$ ; ext. belladonna, gr.  $\frac{1}{4}$ ; ext. hyoscyamus, gr.  $\frac{1}{4}$ ; sulphur, gr. ij; purified aloes, gr.  $\frac{1}{4}$ ; aloin, gr.  $\frac{1}{4}$ . A good combination is aloin, gr.  $\frac{1}{4}$ ; ext. belladonna, gr.  $\frac{1}{4}$ ; ext. nux vomica, gr.  $\frac{1}{12}$ ; ol. theobrom., gr. x. In obstinate cases this may be used night and morning, and later at night only. After some improvement has occurred the aloin may be omitted. Many of the proprietary suppositories contain the ingredients mentioned, particularly belladonna, the dose of which is often considerably larger than should be given. Suppositories are chiefly useful when the trouble is in the rectum and lower colon;

but very little is to be expected from them when it is higher in the intestine.

*Enemata.*—These should be restricted to cases in which only temporary relief is desired. An injection of an ounce of sweet oil may facilitate the passage of very hard and dry stools, and a regular nightly repetition of this, or a somewhat larger amount, for several weeks will often break up a constipated habit. Injections of soap and water may be used to soften hard faecal accumulations. For immediate effect an injection of one or two drachms of glycerin in an ounce of water is perhaps the most efficient means at our command. Cases of faecal impaction are rarely met with in children. They are to be managed as in adults, by repeated injections of warm water or of ox-gall, and sometimes by mechanical removal. For continuous use enemata of water are not to be advised, for larger and larger quantities are required to produce the effect.

*Medicinal Treatment.*—This is the least important part of the management of chronic constipation. No plan is worse than to give some active purgative every third or fourth day and trust matters to take care of themselves the rest of the time. The most valuable drugs are stimulating laxatives, such as cascara, nux vomica, belladonna, hyoscyamus, and phenolphthalein. These are particularly useful in atonic constipation associated with rickets and following diarrhoeal diseases, but they are valuable in all cases. With most drugs the prolonged use of small doses is better than the occasional use of large ones. Calomel is indicated in cases attended with dry, very white stools and marked flatulence; one-fourth to one-half grain of the tablet triturates may be given for two or three successive nights in conjunction with other means. Cascara may be used either in the form of the elixir, dose from one-half to one drachm, or the fluid extract, from one to five drops. Rhubarb, either in the form of the syrup or the mixture of rhubarb and soda, may be given occasionally, but it is not adapted to continuous use. Of salines, magnesia and phosphate of soda are best for continuous use in infants. All the preparations of malt possess slight laxative properties, and are useful in conjunction with dietetic and other medicinal means; any of the extracts of malt may be employed. Castor oil should seldom be given for chronic constipation. Olive oil is often of assistance in the treatment of the constipation both of infants and older children. To the former the usual dose is one teaspoonful three times a day; to the latter, two or three times this amount should be given. Agar-agar by rendering the faecal mass softer and more easily expelled frequently proves a most effective remedy for older children. It should be broken up into fine fragments and mixed with the cereal when eaten or it may be cooked with it. The dose is three or four teaspoonfuls.

## HYPERTROPHY AND DILATATION OF THE COLON

*(Hirschsprung's Disease).*

It is probable that in many cases of chronic constipation, especially among rachitic infants, a considerable degree of dilatation of the colon occurs. However, it seems to be but a temporary condition, disappearing by the third or fourth year.

There is another form of dilatation which may be permanent and is generally believed to be of congenital origin; it is associated with a marked degree of hypertrophy of the muscular walls of the colon. Cases have been observed both in infants and in older children. The prominent symptoms are two: obstinate constipation, which in most of the cases has continued from early infancy, and is sometimes so severe that the patients have gone for two weeks without a movement of the bowels; and distention of the abdomen, which may be extreme, but which may disappear and the abdomen become perfectly flat after the fæces and flatus have been discharged. There is usually emaciation, and from time to time there may be diarrhoea. Death may occur in infancy, or the patients may live to adult life.

In the cases which have come to autopsy there has been found an enormous dilatation of the large intestine, chiefly of the transverse colon and the sigmoid flexure. In one reported case, in a boy of three years, the colon was four inches in diameter, and held fourteen pints of water. In none of the cases was there stricture at any point. The mucous membrane has almost invariably been found ulcerated, this clearly being a secondary process. The muscular walls have been greatly hypertrophied. Medical treatment is palliative only. An artificial anus has been made in several cases with at least temporary benefit. The complete removal of the large intestine has also been performed for this condition.

## INTUSSUSCEPTION.

Intussusception consists in the invagination of one portion of the intestine into another. It occurs most frequently in infancy, being at this age the most common cause of acute intestinal obstruction. The accident is not a common one, but the life of the patient generally depends upon its prompt recognition.

**Varieties.**—Usually the upper part of the intestine is invaginated into the lower, although the reverse is occasionally seen. Intussusceptions may occur at any point in the intestinal tract. Those of the small intestine are called *enteric*; those of the colon, *colic*; and those occurring at the ileo-cæcal valve, *ileo-cæcal* (Fig. 65). Of 90 cases under ten years of age, in which the variety was determined by autopsy or operation, 75 were ileo-cæcal, 9 colic, and 6 enteric. In the ileo-cæcal form a few

inches of the ileum pass through the ileo-cæcal valve, and then invagination of the colon occurs. Cases in which the ileum passes through the valve, but without invagination of the colon, are sometimes classed separately as an *ileo-colic variety*.

*Intussusceptions of the dying*, as they have been called, are met with in my experience in about eight per cent of all autopsies made upon



FIG. 65.—ILEO-CÆCAL INTUSSUSCEPTION.

A specimen removed from a child in the New York Infant Asylum.

infants; they are not often found in children over two years of age. They are descending, enteric, easily reducible, and multiple—usually from eight to twelve invaginations are present. They are more frequently in the jejunum than in the ileum. They usually involve but two or three inches of the intestine, but may include ten or twelve inches. They are found in autopsies upon patients dying of all varieties of disease, and are probably produced in the death agony. Such intussusceptions are without symptoms, and are of no clinical importance.

**Etiology.**—Of 358 collected cases under ten years, the following are the ages reported: under four months, 28 cases; from four to six months, 113; seven to nine months, 71; ten to twelve months, 18; one to two years, 32; two to ten years, 96. Three-fourths of the cases which occur in childhood are, therefore, in the first two years, and one-half of them

between the fourth and ninth months. The greater frequency in infancy is attributed to the thinness of the intestinal walls, the greater mobility of the cæcum and ascending colon, and the presence of other intestinal derangements at this age.

Males are more often affected than females. Of 268 cases in which the sex was mentioned, there were 174 males and 94 females. For this fact there is no explanation. The exciting causes of an attack are extremely obscure. The great majority of cases occur in children who were apparently in perfect health. Some previous intestinal disorder was present in about three per cent of the cases I have collected—diarrhœa, dysentery, colic, chronic indigestion, and constipation, all being mentioned. In four cases the intussusception was ascribed to injury of the abdomen. The association with the general diseases is too infrequent to be of any importance.

**Lesions.**—Nothnagel's animal experiments have shown conclusively that intussusceptions are formed by the irregular action of the muscular walls of the intestine. They can be produced or released at will by varying the application of the electrical current. In the artificial intussusception there is first a contraction of a certain part of the intestine, and if this ceases abruptly the normal gut below this point turns upward and folds over upon the contracted portion, thus forming a minute intussusception (Fig. 66, A). When once begun, the intussusception increases solely at the expense of the external layer (Fig. 66, B). Thus, while the apex of the tumour D remains unchanged, the part of the sheath at A passes to B and then to C, so that the lower part of the intestine is drawn over the upper, rather than the upper crowded into the lower. The mechanism of the invagination was apparently the same when a part of the intestine was first paralysed by crushing, as in the cases in which a spasm of the intestine was first produced.

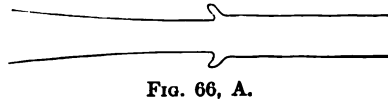


FIG. 66, A.



FIG. 66, B.—MECHANISM OF INTUSSUSCEPTION. (Treves.)

There is little doubt that pathological intussusceptions are produced in the same way as in these experiments. As the invagination takes place, the mesentery is drawn in with the bowel, and always lies between the sheath and the inner layer. To allow intussusception to occur, the mesentery must be unduly long, stretched, or lacerated. Its attachment to the spine causes the intussusception to describe an arc of a circle, the concavity of which is always toward the spine. It also causes a puckering of the tumour. Invagination does not necessarily produce either ob-

struction or strangulation, but usually both are present, and are the chief causes of the symptoms. Traction upon the mesentery leads to obstruction in its vessels, causing congestion, œdema, hæmorrhages, and even gangrene. Obstruction is chiefly due to swelling. It may be due to dragging of the mesentery, which brings the apex of the tumour against the side of the gut, or to bending of the intussusception. Intussusception is usually of all the coats of the intestine. I have, however, seen one, the exact nature of which was determined by operation, in which only the mucosa and submucosa were involved. The invagination was at the ileo-cæcal valve. The symptoms were characteristic except for the absence of tumour.

The great cause of irreducibility in the first two or three days is swelling. I have several times seen at autopsy or operation the intussusception easily reduced, except the last two or three inches of the cæcum or ileum, which was swollen to the thickness of from a fourth to half an inch. Adhesions may prevent reduction, but rarely before the fourth day; they are often absent as late as the sixth or seventh day. They are usually between the internal and middle layers of the intussusceptum, and are due to local peritonitis. In chronic cases, however, they form the principal obstacle to reduction. Other causes of irreducibility are twisting of the tumour and pinching of the prolapsed intestine, especially of the ileum by the ileo-cæcal valve.

Gangrene and sloughing of the gangrenous portion of the intestine occur much more often in acute than in chronic cases. Portions of intestine were passed *per anum* in 24 of 362 cases under ten years, or about six per cent; but only two of these were in infants. Toward the end of the second week is the time when the separation of the sloughs is to be looked for. The amount of intestine discharged varies from a few inches to several feet. Two cases are on record in which the entire colon was passed, the patients recovering, but dying several months later from other causes. At the autopsies the ileum was found attached to the lower part of the rectum just above the anus. In acute cases gangrene occurs about the upper end of the tumour, and the intestine usually comes away in one large mass. In chronic cases shreds of intestine may be discharged for several weeks.

**Symptoms.**—The clinical picture of a case of intussusception is a striking one, and when acute the symptoms are so uniform that, once seen, it can scarcely be overlooked a second time. The patient, usually between six and twelve months of age, is taken suddenly ill with severe pain and vomiting; the pain recurs paroxysmally every few minutes, and the vomiting being first of the contents of the stomach, and afterward bilious. There may be one or two loose faecal stools, then only blood or blood and mucus are passed without any admixture of fæces. The general symptoms are those of great prostration, or even collapse—

pallor, feeble pulse, apathy, and normal or subnormal temperature. The abdomen is relaxed. A tumour is present in the left iliac fossa, or it may be felt *per rectum*. Later there is tympanites; the vomiting and pain continue; there is a steady increase in the prostration, and toward the end a rapidly rising temperature which may reach 105° or 106° F. before death occurs from collapse. If the symptoms continue longer the signs of peritonitis are added. In subacute cases the onset is less abrupt, and pain, vomiting, and constipation less constant and less severe; but the same symptoms are present. In chronic cases the onset is with vague, indefinite intestinal symptoms; pain, vomiting and bloody discharges are usually wanting; there is progressive wasting and more or less diarrhœa, but only the presence of the tumour leads to the recognition of the condition.

In subacute or chronic cases the diagnosis is much more difficult. The general symptoms may be wanting entirely. Vomiting is usually absent; constipation is less marked and there may be none. The only diagnostic feature is the presence of the tumour, usually accompanied by evidences of catarrhal colitis, discharge of mucus, etc.

*Onset.*—Of 193 cases under ten years in which data upon this point could be obtained, the onset was sudden in 181 and gradual in 12 cases. By far the most frequent symptoms of onset are pain and vomiting. In a smaller number of cases the initial symptom is diarrhœa or a discharge of blood and mucus.

*Pain.*—This is rarely continuous, but is intermittent, recurring in paroxysms like those of ordinary colic, but of great severity. No pain in infancy is to be compared with it. The child sometimes shrieks so as to be heard all over the house. Pain is a prominent symptom in over three-fourths of the cases, and is very rarely absent. It is generally more marked for the first two days, but may continue throughout the attack. In a few cases the pain is localised, being usually referred to the region of the umbilicus.

*Vomiting* is more marked at the onset, but may continue throughout the attack. Like the pain, it is more frequent in the acute cases. It is due to intestinal obstruction. Vomiting is present in fully four-fifths of all cases. Usually it is persistent and often projectile. If food is given, vomiting often occurs as soon as it reaches the stomach. Stercoraceous vomiting occurs in about fifteen per cent of the cases in children under ten years, but is not common in infancy. It is rarely present before the third or fourth day. Although a bad sign, it is not by any means a fatal one, as nearly one-half the cases in which it has been noted have recovered; it is to be regarded as indicating complete intestinal obstruction rather than strangulation.

*Tumour.*—This is one of the most important symptoms for diagnosis because of its frequency and its peculiar character. It is present early in



the disease, often in a few hours after the initial symptoms. The following table shows the frequency with which a tumour was present in the different varieties, and the position which it occupied in each. The anatomical variety was determined either by autopsy or operation.

*The Relation between the Tumour and the Different Varieties of Intussusception in 188 Cases under Ten Years.*

| SEAT OF TUMOUR.         | SEAT OF INTUSSUSCEPTION. |             |        |          |             | Total. |
|-------------------------|--------------------------|-------------|--------|----------|-------------|--------|
|                         | Ileo-cæcal.              | Ileo-colic. | Colic. | Enteric. | Not stated. |        |
| Region of cæcum .....   | ..                       | 3           | ..     | 1        | 7           | 11     |
| “ “ ascending colon ..  | 1                        | ..          | ..     | ..       | 12          | 13     |
| “ “ transverse colon .. | 3                        | ..          | ..     | ..       | 13          | 16     |
| “ “ descending colon .. | 3                        | ..          | ..     | ..       | 18          | 21     |
| “ “ sigmoid flexure ..  | 4                        | ..          | 1      | ..       | 8           | 13     |
| Rectum .....            | 25                       | 1           | 7      | ..       | 28          | 61     |
| Protruding from anus .. | 9                        | ..          | 1      | ..       | 12          | 22     |
| Umbilical region .....  | ..                       | ..          | ..     | 1        | ..          | 1      |
| Movable .....           | ..                       | ..          | ..     | 1        | 2           | 3      |
| Site unknown .....      | 1                        | ..          | ..     | ..       | ..          | 1      |
| Total .....             | 46                       | 4           | 9      | 3        | 100         | 162    |
| No tumour felt .....    | 10                       | 2           | ..     | 1        | 13          | 26     |

Tumour was thus made out during life in eighty-six per cent of the cases; and in the great majority of these it was discovered at the first careful examination.

It will be noted that in one-half of the cases the tumour was either felt in the rectum or protruded from the anus, and that in over two-thirds it had advanced as far as the descending colon or beyond. The tumour may reach the rectum in a surprisingly short time, even when the invagination begins at the ileo-cæcal valve. In one of my own cases it was felt in the rectum in less than twelve hours from the onset. The usual description, “sausage-shaped,” is accurate when the invagination is large, the tumour then being from four to six inches long and about an inch and a half in diameter. It is often curved.

During manipulation, or during an attack of pain, the tumour may become more prominent and may be distinctly erectile. To the touch the rectal tumour closely resembles the os uteri, the central opening being the apex of the intussusception. When protruding from the body, the tumour is rarely more than two inches long. It is usually of a deep purplish colour, and may be gangrenous. It has been mistaken for prolapsus ani, polypus, and even hæmorrhoids. In a case which came subsequently under my observation, the tumour was discovered by the mother before the physician had suspected the condition.

*Condition of the Bowels.*—Bloody stools are a very constant symptom. Of 186 cases under ten years in which this condition of the bowels

was noted, blood in the stools was present in seventy-six per cent. There are very often two or three thin, diarrhoeal movements, and then only blood and mucus are passed with no trace of fæces and with no fæcal odour. The amount of blood varies from a quantity sufficient to stain the mucus, to an ounce of semi-fluid blood. It rarely occurs without some mucus. Such discharges frequently follow attacks of severe colicky pain, and may occur several times in an hour. They may continue, or after a day or two they may be succeeded by absolute stoppage. Diarrhoea throughout the attack is rare in children, particularly so in infants. It belongs generally to chronic cases. Constipation is complete in most of the acute cases, neither gas nor fæces being passed; a fact which the discharge of blood and mucus may lead one to overlook.

Tenesmus is very common if the tumour is rectal. Relaxation of the sphincter is met with in a considerable proportion of the cases when the tumour is in the sigmoid flexure, or rectum.

During the first twenty-four or forty-eight hours the abdominal walls are soft and relaxed, and may even be retracted. Usually there is then little resistance to abdominal palpation. After the second or third day there is usually tympanites; but this does not necessarily mean that peritonitis exists. Localised tenderness is a symptom of some importance when a tumour is absent. Scanty urine has been noted in a few cases, but is of no special value in showing the seat of obstruction.

In the acute cases the general symptoms are very striking. They are the ordinary ones of severe shock—marked prostration, pallor with an anxious expression of the face, general muscular relaxation, cold extremities, cold perspiration, and often a subnormal temperature. Early there is marked restlessness, and even convulsions may occur. Later there are apathy, dulness, and semi-stupor. The temperature during the first twenty-four hours is usually not elevated, and is frequently subnormal. Toward the close of the disease it rises rapidly to 103°, 104° F., or even higher, quite independently of peritonitis. A rapidly rising temperature is always a bad symptom, and usually betokens death within twenty-four hours. Wasting is seen in the chronic cases, and may be quite rapid.

*Course, Duration, and Termination.*—Of 198 cases under ten years, 155 were classed as acute, lasting less than seven days; 33 as subacute, lasting from one to four weeks; 10 were chronic, lasting over four weeks. Nearly all the cases occurring in infancy are acute.

Spontaneous reduction is, without doubt, possible in intussusception. Treves and others are of the opinion that this happens much more frequently than is generally supposed, and that many cases of severe colic are really cases of slight intussusception. There are seen in both conditions the tendency to vomit, the paroxysmal pain, the constitutional depression, and often the sudden cessation of the symptoms, especially

under the influence of opium; but to make a positive diagnosis of invagination in such cases is impossible. Intussusception may be cured spontaneously by sloughing of the invaginated part, the continuity of the intestine being preserved by adhesions. Such a result is rare at all ages, and is almost never seen in infancy.

The most frequent cause of death in acute cases is shock. Peritonitis is not found at autopsy or operation so often as might be expected. In fifty-eight autopsies, it was seen but twenty times, and in seven of these it was limited to the intussusception. In but seven cases was there perforation.

**Diagnosis.**—This usually presents no difficulty in acute cases provided the physician has the condition in mind. The great majority of such cases present nearly all the classical symptoms, viz., sudden onset, recurring colicky pains, frequent vomiting, bloody and mucous stools without faecal matter, general prostration or collapse, and low temperature. The records show that the most common error is to regard the case for the first few days as one of gastro-enteritis or ileo-colitis, the physician's attention being engrossed by the vomiting and bloody stools. Given the other usual symptoms, the presence of the characteristic tumour is conclusive evidence of intussusception. Unless the patient is very much relaxed, a satisfactory examination is possible only under full anaesthesia. In any case of acute intestinal obstruction in infants, intussusception should first be considered. I once saw, in a young infant, strangulated hernia produce nearly every symptom of intussusception except the abdominal tumour. Chronic cases present no diagnostic symptoms except the tumour. In both acute and chronic cases the rectal examination is most important for diagnosis, and often settles the question at once.

**Prognosis.**—The prognosis of intussusception depends upon the age of the patient, upon the variety of the disease—whether acute, subacute, or chronic—and upon the time when proper treatment is begun.

There were collected by Pilz in 1870, 94 cases under one year, the mortality being 84 per cent. Of 135 cases of the same age reported between 1870 and 1891 the mortality was 59 per cent. Results in older children were somewhat more favourable. Formerly recovery was rare, except in cases with sloughing; but with earlier diagnosis and a better understanding of the proper methods of treatment, the mortality has been very much reduced. Combining the figures of Pilz with my own, there are 362 cases with 231 deaths, or 63.5 per cent.

Gibson (New York) has collected reports of 187 operations for intussusception, with a general mortality of 51 per cent; in 126 cases, in which the tumour was reducible, it was but 36 per cent; in 61, in which it was irreducible or gangrenous, it was 80 per cent. The table following gives the mortality in relation to time of operation:

| TIME OF OPERATION. |       | Mortality.<br>Per cent. |
|--------------------|-------|-------------------------|
| First day          | ..... | 37                      |
| Second "           | ..... | 39                      |
| Third "            | ..... | 61                      |
| Fourth "           | ..... | 67                      |
| Fifth "            | ..... | 73                      |
| Sixth "            | ..... | 75                      |

After the second day the chances of success are greatly reduced.

**Treatment.**—The diagnosis of acute intussusception once made, laparotomy should immediately be performed without an hour's unnecessary delay. The results following inflation of the intestine with air and injection with water are too uncertain to be depended upon.

Operation should be looked upon as a measure which, if employed reasonably early, offers a good prospect of success. All statistics show that the result depends more upon the time when the operation is done than upon any other single factor. With earlier diagnosis and more prompt resort to operation, the mortality from acute intussusception has, during the past fifteen years, been steadily falling. In chronic cases, also, laparotomy offers altogether the best chance of success.

## CHAPTER IX.

### *DISEASES OF THE INTESTINES.—(Continued).*

#### APPENDICITIS.

APPENDICITIS is met with at all ages, and is not especially a disease of children. When it attacks those over ten or twelve years of age it does not differ greatly from the types observed in adults. All that will be attempted in this chapter will be a consideration of the peculiarities of the disease as it is seen in children, particularly young children. For a fuller discussion of the disease as a whole the reader is referred to works on general medicine and surgery.

**Etiology.**—Of 1,000 cases of appendicitis personally observed by McCosh, 85 occurred in children between the ages of ten and fifteen years; 51 between the ages of five and ten years, and only 17 under five years; of these but 4 were under two years. Churchman's figures from the Johns Hopkins' Hospital, in a total of 1,223 cases, give only 9 cases under five years, and 50 between five and ten years. In infancy and early childhood appendicitis is, therefore, a relatively rare disease. The youngest cases that have come under my observations were in infants of nine and fourteen months respectively. Goyen's case was in an infant

only six weeks old; Shaw's, seven weeks; Demme's, seven weeks; and Savage's, nine weeks old. The predominance of the male sex holds true even in childhood. Of 101 cases under fifteen years, 72 were males and 29 were females.

Regarding the exciting cause of an attack but little is yet definitely known. In only a very small proportion of the cases is a foreign body discovered in the appendix. In one of my own a pin was found, and a number of similar cases are on record. There is, however, almost invariably a faecal concretion which is moulded into the shape of a foreign body, and formerly was often regarded as such. This probably has some relation to the attack by causing disturbances of circulation and increasing the chances of infection. Still and others have called attention to the frequent occurrence of pin worms in the appendices of young children. There is abundant reason for believing that these may at times be the exciting cause of an attack. The bacteria most frequently found in abscesses from appendicitis are streptococci, usually associated with colon bacilli.

**Lesions.**—All the common varieties of acute appendicitis, the catarrhal, suppurative, and gangrenous, are met with in children; and, much less frequently, the chronic form. The lesions present few peculiarities in early life except that, owing, possibly, to the relation of the appendix to the omentum, perforative inflammations are less likely to be circumscribed by inflammatory products and much more likely to result in a general peritonitis than in adults. Whether or not this be the correct explanation, it is certainly true that general peritonitis is a much more common sequel than in adults. Another point of some importance is the fact that in early life the appendix is rather more frequently found out of the usual position. The inflammation excited by pin worms is usually a superficial one; perforation and abscess formation are almost unknown when they are the cause.

**Symptoms.**—In many of the cases the familiar symptoms of appendicitis—vomiting, localised pain and tenderness, muscular rigidity, abdominal distention, and fever—are all present, and the diagnosis is easy. But in perhaps the larger number the disease is irregular in its onset, insidious in its course, and presents at times great difficulties in diagnosis. This is particularly true of appendicitis in children under five years. Vomiting is probably the most constant symptom; it is seldom absent, and usually persistent. If accompanied by pain and constipation, appendicitis should at once be thought of. Pain, though usually present, is often indefinite; it is generally hard to localise and difficult to interpret. It may be referred now to one and now to another part of the abdomen. Often the only evidence of pain is restlessness, irritability, and, in infants, frequent crying. Tenderness is even more difficult to elicit than pain. Young children, especially if nervous and sensitive, shrink from

any touch, and the results of abdominal palpation may be most unreliable. In others of a different temperament positive information may be obtained. In any child under three years, it is practically impossible to make out localised tenderness. The same is true of muscular rigidity. Only with the greatest amount of tact and by diverting the patient's mind, can any information be derived from this part of the examination. Tenderness and muscular rigidity are sometimes shown by the child's disinclination to move either the trunk or lower extremities and by evidences of pain when he is moved by mother or nurse. When associated with vomiting, fever, and constipation, such symptoms are always suggestive.

Constipation is usually present, but by no means so regularly as in adults. Diarrhoea is not at all uncommon, and, when associated with vomiting, tends to divert attention from the appendix to an ordinary gastro-intestinal attack. Abdominal distention, when present, is of much importance, taken with other symptoms. Fever is rather more apt to be high than in adults. But there are many exceptions, and, on the whole, the temperature is a very untrustworthy guide either to diagnosis or prognosis. The leucocyte count is of much assistance in diagnosis, at least in suppurative forms of appendicitis. A leucocytosis of at least 10,000 to 20,000 is usually present, with a polymorphonuclear percentage over 75. Some special symptoms may be seen in appendicitis which are quite misleading. I have on several occasions seen frequent micturition and other marked manifestations of vesical irritation, owing to the position of the appendix behind the bladder. The rigidity of the thigh flexors seen in cases of appendicitis, which come on with subacute symptoms, may give rise to lameness strongly suggestive of hip disease; cases of this kind are not infrequently seen at the Hospital for Ruptured and Crippled.

*Course of the Disease.*—A certain number of cases begin with definite symptoms—pain, vomiting, fever, and constipation—and continue with slowly or rapidly advancing symptoms to increasing prostration, continued vomiting, constipation, rapid pulse, abdominal distention, and rigidity, higher temperature, and death by general peritonitis at the end of five or seven days' illness. Others, with a similar onset, show a gradual abatement of all acute symptoms after a few days, and recovery at the end of ten days or two weeks, followed, perhaps, by another attack after a few months. These types are seen in children as in adults. But others are quite common. A child may be taken ill, sometimes abruptly, sometimes more gradually, with vomiting, which is repeated several times in a single day, afterward only occasionally. There is some pain; it is not very definite and not localised. The prostration is only moderate, the temperature not over 100° or 100.5° F. The examination shows little. Tenderness can not be definitely made out; the child

is irritable, fretful, wishes to be left alone, and resists all efforts at abdominal palpation. The bowels are constipated, or they may be at first loose and afterward constipated. The child does not seem very sick. The attack is probably regarded as an ordinary one of acute indigestion. But things do not improve as they ought. The pulse becomes more rapid, the prostration greater, and the child begins to look seriously ill, though the temperature has not risen. The abdominal distention is now considerable and tenderness undoubted. An operation is decided on, and there is found a gangrenous appendix and a diffuse general peritonitis. Sometimes the grave symptoms develop with great rapidity in the course of a few hours, when previous symptoms had all been mild; sometimes so insidiously that the transition is almost imperceptible.

**Prognosis.**—The prognosis in young children is not good; of 132 collected cases in infants and very young children the mortality was 38 per cent. But in those over seven years old the outlook is rather better than in adults. The results depend much upon early diagnosis and proper treatment. General peritonitis, it is generally agreed, occurs much oftener in children than in adults; it is the cause of death in about 80 per cent of the cases. Of 43 fatal cases, nearly all of them from general peritonitis, only 6 died during the first three days, 19 from the fourth to the seventh day, 13 in the second week, and 5 in the third week. If general peritonitis occurs, the chances of recovery after operation are, however, better with children than with adults.

**Diagnosis.**—The diagnostic symptoms of appendicitis are a sudden onset with vomiting, sharp pain in the abdomen, and persistent acute local tenderness in the right iliac fossa. Rigidity of any or all of the abdominal muscles is a less significant sign. Constipation is more frequent than diarrhoea, though the latter is not rare. There is almost invariably some elevation of temperature, but not often high fever.

Appendicitis may be confounded with acute indigestion, and in infants with enterocolitis; in older children with abscesses due to pyloric ulcer, or with typhoid, by the absence of localized tenderness and fever, by a short duration, and by the fact that the pain is generally less intense. Nevertheless, fever in a child over three years old should, however, always be regarded with suspicion. From acute indigestion the diagnosis of appendicitis is difficult at the onset, and it may be impossible to identify the disease. However, the pain of indigestion is usually severe when the fever is usually absent. It should be remembered that the pain of appendicitis is not always localised, nor is the tenderness confined to the right iliac fossa. If the presence of pain, vomiting, prostration, tenderness, and the general signs of the constitutional disturbance are not overlooked, it is not so often known the truth as in the case of the adult, in whom the signs are more likely to be mistaken for indigestion. With this caveat, the vomiting, acute localised pain, and

sometimes also localised tenderness. Cyclic vomiting is distinguished by the history of previous attacks, the greater frequency with which the vomiting occurs, its abrupt cessation after twenty-four to forty-eight hours, the sunken abdomen, and the absence of pain, tenderness, and rigidity. The presence of early acetonuria is also characteristic. Intussusception, with its pain, colic, and vomiting, may suggest appendicitis, but is very rare, except in infants. Fever is absent early in the disease, and a tumour is usually present. Acute or subacute suppuration in the right iliac fossa is almost invariably due to appendicitis.

The leucocyte count may be of considerable assistance in differentiating appendicitis from colic, cyclic vomiting, ileo-colitis, and intussusception. It should, however, be remembered that in some of the gravest cases the leucocytosis may be slight or there may be none at all. On the whole, while the presence of marked leucocytosis—i. e., above 20,000—may be of considerable assistance in the diagnosis, no inference can be drawn from a normal count or a slight leucocytosis if the child is greatly prostrated. Whenever, in children over two years old, there are symptoms pointing to acute peritonitis, no matter what their combination or variety, appendicitis should always be suspected.

**Treatment.**—Absolute rest in bed can not be too strongly insisted upon whenever appendicitis is suspected, no matter how mild the attack may appear. As a local application, the ice-bag is to be preferred. Opium should not be given. It does harm by obscuring important symptoms and increasing constipation. The colon should be kept empty by the daily use of enemata. After a thorough clearing of the bowels in the beginning, preferably by a saline, cathartics are to be avoided.

Appendicitis is a surgical disease, and surgical advice should be sought early. In deciding as to the time of operative interference, it should be remembered that the natural course of the disease in children is much less likely to be favourable than in older patients; that the dangers of general peritonitis are much greater; that the progress of the disease is much less regular; that grave conditions are not revealed at once by grave symptoms; that the disease is an insidious one, and that to foretell the outcome even in the mildest cases is impossible. Taking all these things into account, I believe that immediate operation, once the diagnosis is made, is the course to be recommended in all cases of acute appendicitis in children. The younger the child the greater the urgency for operation.

#### INTESTINAL WORMS.

Judging by published reports, intestinal worms are much more common in Europe than in this country. In 10,000 patients treated for medical diseases in my dispensary service, there was positive evidence of worms in but 79 cases. Of these, 9 had tapeworms, 40 roundworms, 27



threadworms, and 3 both round and threadworms. In private practice among the better classes, worms are certainly rare.

**Cestodes—Tapeworms.**—Cestodes are usually introduced into the body by the ingestion of some form of food containing larvæ (cysticerci). The larva of the *tænia solium* is most frequently found in pork; that of the *tænia mediocanellata* in beef; that of the *bothriocephalus latus* in fish; that of the *tænia cucumerina* inhabits dog or cat lice, being introduced into the intestinal tract accidentally by the hands. Several varieties of *tænia* are found in the human intestine.

**TÆNIA SAGINATA OR MEDIOCANELLATA—BEEF TAPEWORM.**—Infection results from eating raw or partially cooked beef containing cysticerci. The worm is from twelve to twenty feet in length, and has a square pigmented head without hooks but provided with four suckers. The full-sized segments are from one-half to three-fourths of an inch long and about half as wide.

**TÆNIA SOLIUM—PORK TAPEWORM.**—This is a rare form in children, and comes from eating raw or partially cooked pork or sausage. It is from six to ten feet in length, the segments being nearly square. The head is about the size of a mustard seed and is pigmented. It also is provided with four suckers and a proboscis, surrounding which is a circle of about twenty-six hooklets.

**TÆNIA CUCUMERINA OR ELLIPTICA.**—The larvæ of this form develop in a louse found on the skin of dogs and cats. Children who play with infected animals are the ones affected, the parasite being conveyed to the mouth usually by means of the hands; it may thus be found even in young infants. This form of *tænia* is much smaller than either of the preceding varieties, the full length being only from six to twelve inches.

**BOTHRIOCEPHALUS LATUS.**—This is a rare form except in the sea countries of northern Europe and Switzerland, where it is said to be very common. The larvæ are harboured by certain fish, through which they are introduced into the body. The full-grown worm is from twenty-five to thirty feet in length.

**TÆNIA NANA.**—The *tænia nana*, or dwarf tapeworm, is the smallest of all the cestodes. It is a narrow worm of one-half to three-fourths of an inch in length, and is composed of one hundred to two hundred segments. It has a slender neck and globular head which contains four suckers and twenty or thirty hooklets. The habitat of the *nana* is the upper part of the ileum where it is often found in immense numbers. A single stool may contain several hundred worms. The ova have two definite membranes within the inner one of which three pairs of hooklets are found. The cysticercus stage of this parasite is not known. It is probable that infection occurs from swallowing the ova themselves. As a similar parasite inhabits the intestinal tract of rats and

mice it is possible that these animals play a part in transmission. From the observations of Schloss it seems probable that in the vicinity of New York this is the most frequent intestinal parasite of childhood.

**SYMPTOMS.**—The only positive evidence of tapeworm is the discharge of the worms or separated segments, either singly or in groups. Occasionally worms pass into the stomach and are vomited. Various abdominal symptoms may be associated with worms, but most of these are very indefinite in character and are more often due to other causes. The most frequent symptoms are bad breath, various annoying sensations, colicky attacks, inordinate or capricious appetite, and diarrhœa. Usually, if the patient is in good health, no constitutional symptoms are seen. Sometimes, particularly with the *bothriocephalus latus*, there is a very grave degree of anæmia. The increase in the number of eosinophile cells in the blood is of considerable diagnostic value. They generally form from four to ten per cent of the leucocytes, while in normal blood the usual number is less than two per cent. Many cases are on record, some of them in children, in which the symptoms of pernicious anæmia have been present and have disappeared after the expulsion of the tapeworm. Nervous symptoms are not so often seen as with round-worms, and will be discussed in connection with them.

**TREATMENT.**—Prophylaxis requires the cooking of meat to a sufficient degree to destroy the cysticerci. There is especial danger in eating raw pork or sausage; that from rare beef is much less. The list of drugs used for the expulsion of the worm is a long one; probably the most efficient is the oleoresin of male fern; it is, however, difficult to administer and it is very likely to provoke vomiting. It may be given in capsules containing ℥x to ℥xx, or in an emulsion made up with simple elixir and acacia, in which ℥v to ℥x are contained in one drachm. For a child of four years at least one drachm of the male fern should be given in the course of six to eight hours. The vermifuge should be preceded by several hours' fasting, and the bowels previously opened by a laxative. The following plan of administration has been found satisfactory: A light supper of milk, and in the morning a saline laxative on rising, but no breakfast; after the saline has acted freely the remedy is to be given, and following the last dose, half an ounce of castor oil or some other active purge. The effect of the cathartic is aided by a large injection of warm soap and water. Only milk should be given that day. The fragments passed should be carefully examined to see if the head has been expelled, as the worm is very likely to be broken at the neck. If this occurs it will grow again, and in about three months segments will appear in the stools. Other drugs useful for *tænia* are pumpkin seeds which are given in powdered form, infusion of pomegranate root, turpentine, and chloroform.

**Nematodes.**—Three varieties are found in the intestinal canal, the *ascaris lumbricoides*, the *oxyuris vermicularis*, and the *uncinaria Americana*.

**ASCARIS LUMBRICOIDES—ROUNDWORM.**—This worm is usually found in the small intestine. It is much more frequently met with in children than is the tapeworm. It is exceedingly rare in infancy, but is usually seen between the third and tenth years. In over one thousand autopsies upon infants I have only once found a roundworm in the intestine.

The roundworm resembles the ordinary earthworm; it is from five to ten inches long, the female being longer than the male. It is of a light gray colour with a slightly pinkish tint, cylindrical, and tapering toward the extremities (Fig. 67). The eggs are oval in form, about  $\frac{1}{100}$  inch in diameter, and numbered by millions. These worms rarely exist singly; usually from two to ten are present, but there may be hundreds. When very numerous they coil up and form large masses, which may cause intestinal obstruction.

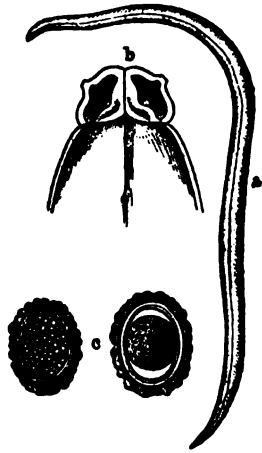


FIG. 67.—*ASCARIS LUMBRICOIDES*. a, entire worm; b, head; c, eggs. (Jaksch.)

The migration of these worms is curious, and in some instances truly remarkable. They frequently enter the stomach and are vomited. Occasionally one may appear in the nose. They have been known to pass through the Eustachian tube into the middle ear and to appear in the external meatus. Entering the larynx they have produced fatal asphyxia. It is not very rare for them to enter the common

bile duct and produce jaundice. They may even enter in great numbers the smaller bile ducts and produce hepatic abscesses. They have been found in the pancreatic duct, in the vermiform appendix, and in the splenic vein. It has long been known that they would perforate an intestine which was the seat of ulceration, but well authenticated cases have been reported in which they have perforated an intestine previously healthy, setting up a fatal peritonitis. In Archambault's case they perforated the stomach. In cases of a persistent Meckel's diverticulum, worms have been discharged from an umbilical fistula. They have been found in umbilical abscesses. Considering, however, the frequency of roundworms, migrations are rare.

**Symptoms.**—The symptoms of roundworms are of the most indefinite kind; often there are none until the worm is discovered in the stools. It is then fair to assume that other worms are also present. The most frequent abdominal symptoms are colic, tympanites, and other symptoms of indigestion, loss of appetite, restless, disturbed sleep, grind-

ing of the teeth at night, and picking the nose. These symptoms are much more frequently due to other causes than to worms, but when all are present the existence of worms should be suspected.

A great variety of nervous symptoms may be associated with intestinal worms. They are more often seen with lumbricoids than with either of the other varieties. The symptoms may be of the most puzzling character, and may simulate very closely those of serious organic disease. There may be prolonged low fever, chills, headache, vertigo, hallucinations, hysterical seizures, epileptiform attacks, convulsions, tetany, transient paralyses such as strabismus, and even hemiplegia and aphasia. All these have been observed in connection with intestinal worms, and from the fact that the symptoms disappeared completely after the worms were expelled there seems to be but little doubt that they were the cause of the symptoms. As in the case of the abdominal symptoms, however, intestinal worms are only one of the causes of such nervous disturbances, and certainly not the most frequent; but the possibility that nervous disturbances may depend upon worms should not be overlooked. The blood generally shows eosinophilia, as in patients with tapeworm.

The only positive evidence of the existence of roundworms is the discharge of a worm from the body, or the discovery of the ova in the stools. A microscopic examination of the stools is a valuable means of diagnosis, and one that is too infrequently employed. When worms are present the ova may be found in great numbers. Their continued presence, after the discharge of one worm, indicates that other worms remain.

*Treatment.*—Altogether the most efficient agent for the removal of the worms is *santonin*. The same plan of administration may be followed as in the case of the tapeworm, viz., to give the drug on an empty stomach, preceded by a laxative. *Santonin* is best given in powdered form mixed with sugar. For a child of five years as much as three grains are usually required. This amount should be given in three doses at intervals of four hours, soon followed by a purge of calomel or castor oil. Or one or two grains of *santonin* may be given with half the amount of calomel every other night for three or four nights. The great difficulty with *santonin* is its tendency to provoke vomiting. Occasionally in susceptible children, even with ordinary doses, toxic symptoms may develop, such as yellow vision, dark red or yellow urine, and nervous excitement or delirium.

**OXYURIS VERMICULARIS—PINWORM—THREADWORM.**—The *oxyuris* (Fig. 68) resembles a short piece of white thread. The female is about one-third of an inch long, the male about one-half that length, but is less frequently seen. The worm tapers toward the tail. The ova are of slightly irregular size, and are considerably smaller than those of the roundworm.

The *oxyuris* inhabits the rectum, the cæcum, and, according to Still,

very frequently the appendix. These worms may be found also in the lower small intestine, in the stomach, and even in the mouth. If present in the rectum they are usually discovered by separating the folds of the anus. The number of worms is usually large. The irritation to which

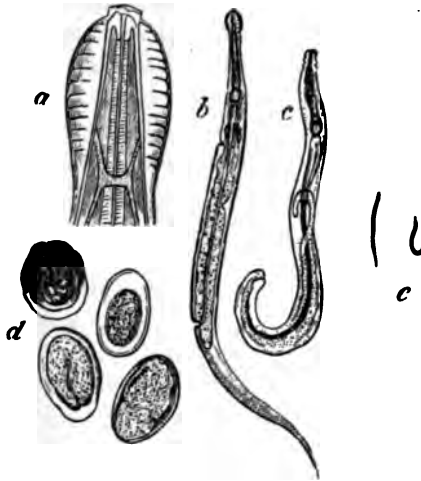


FIG. 68.—PINWORMS. *a*, head; *b*, female; *c*, male; *d*, female and male, natural size; *d*, ova. (Jaksch.)

they give rise causes a great production of mucus, and frequently leads to a chronic catarrh of the colon of considerable severity. The worms are imbedded in the mucus; often they form with it small balls. According to Leuckart, they are incapable of multiplying *in situ*. Doubt has recently been thrown upon this view by the observations of Still. From the immature character and the large numbers of the worms found in the appendix (111 in one case), this writer believes that the appendix may be a breeding place. The ova as well as the worms are passed in enormous numbers with the stools. They attach themselves

to the folds of the skin, the hairs about the anus, and even to the genitals. The patient may, through lack of cleanliness of the parts, continually re-infect himself. After discharge from the body, the ova may be carried by flies and deposited upon fruits, vegetables, or in drinking water.

*Symptoms.*—The principal local symptom caused by the oxyuris is itching of the anus or the genitals. This is caused by the migration of the worms from the bowel, and usually comes on at about the same hour at night, generally soon after the patient has retired. It is sometimes so intense as to be almost intolerable. It leads to frequent micturition, to incontinence of urine, in the male to balanitis, and in the female to vaginitis or vulvitis, and in both, but especially in the latter, it may be the cause of masturbation. Owing to the catarrhal colitis which is excited, there is discharged from time to time a large quantity of mucus. Severe colicky pains are often associated. The irritation may lead to prolapsus ani. Nervous symptoms are not so frequently associated as with the other varieties of worms, although I have seen at least one case of chorea in which they were almost certainly the cause. They have been known to excite convulsions. The general health is sometimes undermined and there may be marked and progressive loss in weight.

*Treatment.*—This is usually spoken of as a very simple matter, and no doubt in recent cases, or where the number of worms is small, this is true; but where the number is large, and considerable catarrhal inflammation of the colon is present, it is often a matter of the greatest difficulty to rid the bowel of these parasites. Cases frequently resist treatment by injection for months, even though thoroughly used. The reason for this is, that only the lower colon is reached by injections while the worms may be chiefly in the cæcum and even in the appendix and small intestine. While, therefore, injections are important and indeed invaluable, they can not be relied upon exclusively. The most scrupulous attention to cleanliness is an absolute necessity as the first step in the treatment of all cases. It is well to bathe the parts about the anus after each stool, and even two or three times a day, with a bichloride solution, 1 to 10,000. Itching is best controlled by the application of mercurial ointment to the folds of the anus at bedtime, this effectually preventing the escape of the worms from the bowel. The local application of cold will sometimes have the same effect. The most efficient of the injections is probably the bichloride. The colon should first be thoroughly cleansed by an injection of lukewarm water containing one teaspoonful of borax to the pint, in order to remove the mucus. When this has been discharged, half a pint of the bichloride solution of the strength mentioned should be injected high into the bowel through a catheter, and retained as long as possible. This should be repeated every second or third night. On other nights a simple saline injection may be employed. The infusion of quassia, asafoetida, aloes, and garlic are also useful. Solutions of carbolic acid should never be employed.

When the worms are high in the colon, drugs by the mouth must be combined with injections. Probably the most efficient remedy is *santonin*, which may be used as for roundworms. The expulsion of the worms is aided by saline cathartics; simple bitters, such as gentian and quassia, are also of some value. I have known one case, which resisted for over two years everything which had been tried, to be cured in two or three weeks by injections of a decoction of garlic, in connection with which garlic was given in liberal quantities by the mouth.

*UNCINARIA AMERICANA* OR HOOKWORM.—This belongs to the class of nematodes. The males are one-fourth to one-half inch in length and the females slightly longer. The parasite resembles the *ankylostomum duodenale* of Europe. Infection usually takes place through the skin of the bare feet, more rarely that of the hands. It is possible, however, to contract the disease by eating dirty fruit or vegetables contaminated by the developing larvæ; but infection does not occur from swallowing the ova or young larvæ. After entering the skin the larvæ find their way into the circulation and thus reach the lungs. From the lungs they may migrate or be coughed up into the mouth and then swallowed. They

are not acted upon by the gastro-intestinal secretions, and in the upper part of the small intestine they develop into mature worms. These may exist in the small intestine for years.

The symptoms in the milder cases are minor digestive disturbances, general malnutrition with moderate anæmia and arrested growth. In the more severe cases the anæmia is very marked, the hæmoglobin often falling to thirty per cent or below. The leucocytes are normal in number or slightly increased; but the percentage of eosinophiles is above the normal. Usually the proportion reaches five or ten per cent; it may however be twenty-five per cent or even higher. Oedema of the face is common and there may be general dropsy without albuminuria. Affected children besides being very backward in physical development, are dull, inattentive and entirely wanting in physical or mental energy. The appetite is sometimes absent; but more characteristic is the craving, not only for every kind of food, but for such articles as clay, dirt, chalk, etc. Death may be due to a progressive failure of nutrition or to intercurrent disease.

Prophylaxis in the individual consists chiefly in the protection of the feet of persons living in an infected district, by wearing shoes. The chief remedy for the hookworm is thymol. Its administration should be preceded by one or more full doses of the sulphate of magnesia or soda given upon a fasting stomach. The quantity of thymol given to a child of five years should be six or eight grains in divided doses in the course of three or four hours. It may be administered either in capsule or in suspension. Two hours after the last dose, the salts should be repeated; but no food should be given until the cathartic has acted freely. Castor oil should not be used. A repetition of the treatment is often necessary before a cure is accomplished.

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## CHAPTER X.

### *DISEASES OF THE RECTUM.*

#### PROLAPSUS ANI.

UNDER this term are included two conditions. In the first, or partial prolapse, there is simply an eversion of the mucous membrane which protrudes beyond the sphincter. In the second, or complete prolapse, there is invagination of the rectal wall for a variable distance, usually two or three inches.

**Etiology.**—Prolapse is most common in children during the second and third years. Its frequency in early life is partly due to the lack of support furnished by the levator-ani muscles. It also occurs very

readily when the ischio-rectal fat is scanty; it is therefore often seen in children suffering from marasmus. The exciting cause may be anything which provokes severe and prolonged straining. This may be either the tenesmus accompanying inflammation of the rectal mucous membrane or chronic constipation. It may come from phimosis or stricture of the urethra, and it is a very frequent symptom of stone in the bladder.

**Symptoms.**—Prolapse usually occurs during the act of defecation. It is generally easily reduced, but shows a great disposition to return with every stool. In obstinate cases the bowel comes down at other times. The appearance of the tumour varies with its size. In the slighter form there is simply a ring composed of a fold of mucous membrane surrounding the anus. In the more severe form there is a flattened, corrugated tumour, usually about the size of a small tomato (Fig. 69). The



FIG. 69.—PROLAPSUS ANI.

mucous membrane covering the tumour is of a deep purplish-red colour, and bleeds readily. It may be the seat of catarrhal or membranous inflammation. The diagnosis in most cases is easy, although the tumour has been confounded with polypus and intussusception.

**Treatment.**—In most cases reduction is easily accomplished by laying the child upon its face across the lap, and making gentle pressure upon the tumour with oiled fingers. The application of cold, either by means of ice or cold cloths, is of assistance in cases which are not at once reduced by pressure. After reduction, in the milder cases the child should be kept upon its back for at least an hour. When the tumour tends to come down with every stool, special attention should be given at this time. If an infant, the bowels should always move while the child lies upon his back, and during defecation the buttocks should be pressed together by a nurse. Older children should use an inclined seat placed at



an angle of about forty-five degrees, but should never sit upon a low chair or assume any position in which straining is easy. After defecation the patient should lie down for at least half an hour. Where there is constipation, the bowels should be kept free by means of laxatives. If there is a diarrhoea, tenesmus may be overcome by frequent sponging with ice water, or by the use of small injections of ice water and tannic acid, in the proportion of twenty grains to the ounce. In more severe cases it may be controlled by the use of suppositories of opium. When the bowel tends to come down frequently, this may be prevented by the use of an adhesive strap two or three inches wide, placed tightly across the buttocks. This is better in the milder cases than a T-bandage. The great majority of the cases are cured by these means in the course of a few weeks.

In the most severe cases the bowel not only protrudes during defecation, but also in the interval, and it may be down for days at a time. Such cases are rarely seen except in infants who have very flabby muscles, and but little adipose tissue at the floor of the pelvis. Reduction is sometimes difficult in cases when the prolapse has lasted a long time. It is often facilitated by painting the protruding part with a two-per-cent solution of cocaine, and then dilating the sphincter by passing the finger into the central opening of the tumour. After reduction, suppositories containing from one-fourth to one grain of cocaine may be inserted. They are more efficient than those containing opium or belladonna. A firm pad should be applied over the anus, held in position by a T-bandage. For several days at a time a short rubber tube may be kept in the rectum, held in place by adhesive plaster. The bowels should be kept freely open. Where all other measures fail, the protruding part may be touched with the Paquelin cautery, linear markings being made at intervals of an inch. Amputation or excision is not required in children.

#### FISSURE OF THE ANUS.

This is not a very uncommon condition in children. The most frequent cause is the passage of a large, hard, fecal mass. Sometimes it results from traumatism inflicted with the nozzle of a syringe while giving an enema. It may be produced by the scratching excited by pin-worms. In the beginning there is a simple tear at the margin of the anus. The laceration which is produced usually heals promptly; but if the cause is repeated, healing is prevented, and there is finally produced a linear ulcer, or a true fissure, which may last for some time and be a source of great annoyance.

A fresh fissure has the appearance of any other tear at a muco-cutaneous orifice. One of longer standing has a gray base, slightly indurated edges, often discharges a small amount of pus, and bleeds a drop or two.

with nearly every movement of the bowels. The most constant symptom is pain, which usually occurs with the act of defecation, and continues for some time afterward. It is most severe when the fissure is just at the margin of the sphincter, and leads the child to resist every inclination to have the bowels move, so that it becomes a cause of chronic constipation, which condition again greatly aggravates the fissure. The pain is often referred to other parts in the neighbourhood.

The treatment is simple and usually efficient. It consists in cleanliness, overcoming the constipation, and touching the fissure with nitrate of silver, preferably with the solid stick. If the case is not speedily relieved by such measures, the sphincter should be stretched as in adult patients.

### PROCTITIS.

Proctitis, or inflammation of the rectum, usually occurs with inflammation of the rest of the large intestine, but it may occur alone. It is to the cases in which only the rectum is involved that the term is generally applied.

The causes are for the most part local. A frequent one in infants is the use of irritating injections or suppositories, either for the relief of constipation or as a means of administering certain drugs. I have seen one obstinate case in an infant a year old, following the prolonged use of glycerin suppositories. It is sometimes caused by traumatism, especially by the careless giving of an enema. It accompanies pinworms. In certain cases it may result from direct infection through the anus. This may be from a gonococcus inflammation extending from the vagina or urethra, or from an infection due to other bacteria, particularly in cases of measles, scarlet fever, and diphtheria; or, finally, it may be due to syphilis. The varieties of inflammation are the same as in the rest of the intestine. Proctitis may thus be catarrhal, membranous, or ulcerative.

**Catarrhal Proctitis.**—The pathological conditions are the same as in ordinary catarrhal inflammation of the intestinal mucous membrane. By the introduction of a speculum, or by simply everting the mucous membrane, it is seen to be reddened, swollen, and bleeds easily. There is a copious secretion of mucus. In cases of long standing there may be superficial ulceration appearing as a white or yellowish-white surface, usually just inside the sphincter.

The symptoms are chiefly local, although a condition of general irritability may result from the local condition. There is heightened reflex action, so that the stool often comes with a squirt. There is pain with defecation, and mucus is discharged, usually as a clear, jelly-like mass, and sometimes in the form of a cast, but not generally mixed with the stool. There are usually traces of blood, sometimes quite large hæmorrhages. In the most acute cases, tenesmus is present both during and

after the stool. There may be prolapsus ani. The skin in the vicinity is irritated by the discharges, most frequently so in infants. If the cause is pinworms, there may be intense itching. The duration of the disease is indefinite, depending upon the cause. It may be a few days or many months. The inflammation may extend from the rectum to neighbouring parts, leading to ischio-rectal abscess.

**Membranous Proctitis.**—It has been customary to describe this as a complication of diphtheria, usually occurring with diphtheria of the external genitals. As very few of these cases have been studied bacteriologically, it is impossible to say what proportion of them, if any, are to be regarded as true diphtheria. It is probable that the great majority are due to infection by streptococci. When the infection is from the intestine above, the rectum is never affected alone. When it is from below, this may be the case. The lesions are the same as in membranous inflammation occurring higher in the colon. The symptoms resemble those of the catarrhal variety, with the addition that the stools contain pieces of pseudo-membrane. This can be made out only by repeatedly washing the discharges with water. If accompanied by prolapse, the pseudo-membrane may be seen. Membranous proctitis may be complicated by a membranous inflammation of the genitals or the perinæum. Although it is usually acute, it may last for weeks.

**Ulcerative Proctitis.**—Ulcers of the rectum may be the result of a catarrhal inflammation; these, however, are usually superficial, affecting the mucous membrane only, and in most cases heal rapidly. Sometimes they extend more deeply into the submucous or even the muscular coat. They are then chronic, often very obstinate, and may last indefinitely. Follicular ulcers of the rectum are nearly always associated with the same condition in the sigmoid flexure. These are always multiple and usually small, rarely being more than a quarter of an inch in diameter. Sometimes the small ones coalesce, producing much larger ulcers. Membranous proctitis is rarely followed by ulceration, although this is a possible result where sloughing has occurred. Single ulcers may be of tuberculous origin. Steffen reports two cases of tuberculous ulcer of the rectum in children of seven months and three years respectively. I have, in a young infant, seen one such ulcer, which was fully three-fourths of an inch in diameter, and was not associated with other tuberculous disease of the large intestine. Syphilitic ulcers are extremely rare in children.

The symptoms of ulcer of the rectum are mainly two—pain and hæmorrhage. The pain is of variable intensity, and may be referred to the coccyx, or to any of the neighbouring parts. The amount of bleeding may be small, the blood coming in clots, or it may be fluid and in so large a quantity as to produce general symptoms. It usually accompanies every stool. In addition the stool contains more or less pus, par-

ticularly in chronic cases. When the ulcer is low down, tenesmus is present and may be a prominent symptom. A positive diagnosis of ulcer can be made only by examination with a speculum.

**Treatment.**—In cases of acute catarrhal proctitis injections of some bland fluid should be employed, such as a starch-water, limewater, a mixture of oil and limewater, or a warm one-per-cent saline solution. The local cause, if one exists, should be removed. The disordered digestion, when present, is to be treated according to its special symptoms. In the most acute cases the patient should be kept in bed. When the tenesmus is severe, suppositories of opium may be used. In the more chronic cases saline injections should be given, and followed by a mild astringent like tannic acid, ten grains to the ounce, or a one-per-cent solution of hamamelis. Cases associated with pinworms are especially obstinate. Here the treatment is first to be directed to the worms, and afterward to the proctitis.

In the membranous cases the same measures are to be employed, and in addition the injection of a warm boric-acid solution two or three times a day.

Cases of ulcer require the most careful treatment. In many there is but little tendency to spontaneous recovery. An examination with the speculum should be insisted upon in all cases of chronic proctitis, to make sure of the diagnosis. Rest in bed is essential to a rapid improvement. The patient should be put upon a bland diet, especially of milk, and the bowels kept freely open by the use of laxatives, and injections twice a day of a saturated boric-acid solution. Locally there should be applied a solution of nitrate of silver, one grain to the ounce, the bowel having previously been washed with tepid water. If a stronger solution than this is used, it should be neutralised after half a minute by the injection of a salt solution.

#### ISCHIO-RECTAL ABSCESS.

This is not a very rare condition even in infancy. Infection from the rectum, usually through the lymph channels, seems to be the most common cause, although sometimes the abscess may be traced directly to traumatism. In a single year I have seen six such cases. All but two were small, circumscribed abscesses, and quite superficial, apparently starting as an acute inflammation of the lymph glands of the region. They are analogous to a similar process in the lymph glands of the neck, seen in infancy. These cases healed promptly after incision. In other instances there is seen a disposition to burrow, as in adults. Only once have I met with diffuse suppuration in the ischio-rectal region, terminating in sloughing and death, and this was in an infant only three months old.

Essentially the same varieties of inflammation are seen in early life as

in adults. Most of these cases recover promptly after simple incision and cleanliness, fistula being a rare sequel.

#### RECTAL POLYPUS.

Polypi are rarely seen in children, but, when present, may be the cause of rather obscure symptoms. The most important one is hæmorrhage. This at first occurs at intervals of days or weeks. The amount of blood lost is from a drachm to an ounce or more. Later, the hæmorrhages become more frequent and may be almost continuous, although rarely profuse enough to produce serious symptoms. The diagnosis of polypus is made only after a local examination. Sometimes the tumours are within the reach of the finger; in other cases a proctoscope must be employed. Spontaneous cure often takes place by the sloughing of the tumour, after which the bleeding soon ceases. In other cases operation is necessary.

#### HÆMORRHOIDS.

These, fortunately, are not often seen in children, although they occur in those as young as three or four years, and in some cases may even be congenital. The principal cause is chronic constipation, rarely diarrhœa. The tumours are generally small and external, the chief symptom complained of being pain on defecation. Bleeding sometimes accompanies the pain, but the hæmorrhages are usually small. The treatment is to be directed toward the underlying cause. In most of the cases this suffices to cure the condition. I have rarely seen in a young child a case requiring operation, although neglect may make this procedure necessary.

#### INCONTINENCE OF FÆCES.

Inability to control the fæcal evacuations is seen in certain cases of paraplegia due to myelitis, in injury of the lumbar portion of the spinal cord, and in spina bifida. It is also seen in acute disease, as in the coma of meningitis, and occasionally in the typhoid condition and in extreme adynamia, from any cause. It is quite common in severe attacks of chorea. In all these conditions incontinence of fæces is a symptom giving rise to much annoyance and needing careful attention. Uncleanliness with reference to excreta, seen in idiocy, can hardly be classed as incontinence.

Besides these familiar forms, the condition is sometimes seen from causes somewhat resembling those of incontinence of urine. The tone of the sphincter becomes so feeble that it does not resist even the slightest impulse to evacuate the rectum. The discharge may take place with but little warning, and may occur either by day or night. In some cases a local cause exists, such as stretching of the sphincter by an old rectal

prolapse. It has followed overdistention of the rectum from prolonged chronic constipation. Ostheimer reports a case in which a vesical calculus was present. It is sometimes seen after severe acute illness, as a result of a loss of general muscular tone. In certain children it has been known to persist from infancy until the age of ten or twelve years. It may come on as a somewhat acute condition in highly nervous patients with poor general nutrition. The causes are chiefly of local and nervous origin. The treatment is rather unsatisfactory, except in recent cases and in those due to local causes which can be removed. If constipation exists the rectum should be emptied daily, preferably by an enema. The remedies which have proven most successful are strychnia, ergot, and belladonna, but they must be given in full doses, sometimes advantageously by suppository as well as by mouth. The general health should receive careful attention.

## CHAPTER XI.

*DISEASES OF THE LIVER.*

ASIDE from the different forms of degeneration which are seen in the various infectious diseases, the liver is not often the seat of serious disease in infancy and early childhood. In later childhood nearly all the forms seen in adult life are occasionally met with, although even then they are quite rare.

**Size and Position.**—The weight of the liver in the newly-born child, from one hundred and seven observations of Birch-Hirschfeld, is 4.5 ounces (127 grammes), or about 4.2 per cent of the body weight. The following table gives the results of one hundred and seventy-four observations upon the liver in infancy in the autopsy room of the New York Infant Asylum:

| Age.               | AVERAGE. |          | Per cent of body weight. |
|--------------------|----------|----------|--------------------------|
|                    | Ounces.  | Grammes. |                          |
| 3 months . . . . . | 6.3      | 180      | 3.1                      |
| 6 " . . . . .      | 7.5      | 212      | 3.0                      |
| 12 " . . . . .     | 11.0     | 311      | 3.40                     |
| 2 years . . . . .  | 14.0     | 397      | 3.37                     |
| 3 " . . . . .      | 16.0     | 453      | 3.26                     |

In adults, according to Frerichs, the weight of the liver is about 2.5 per cent of the weight of the body.

The upper border of the liver is best made out by percussion. In the child, the upper limit of the liver dulness in the mammary line is found

in the fifth intercostal space; in the axillary line, in the seventh space; posteriorly, in the ninth space. The lower border is best determined by palpation. This, as a rule, in the mammary line is found about one-half an inch below the free border of the ribs. According to Steffen, the left lobe is relatively larger in the child than in the adult. The liver may be displaced downward by contraction of the chest, as in rickets, or by an accumulation of fluid in the pleural cavity. It is frequently found lower than normal in conditions of great emaciation, owing to relaxation of the abdominal walls and its ligamentous supports. Upward displacement is much less frequent, and depends usually upon ascites or abdominal tumours.

**Malformations and Malpositions.**—Congenital malformations relate chiefly to the bile ducts. These have been considered in the chapter devoted to Icterus in the Newly Born.

The liver may be found upon the left side in cases of general transposition of the viscera. In diaphragmatic hernia it has been found in the thoracic cavity.

#### CHRONIC FAMILY JAUNDICE.

This disease is usually hereditary, but it occasionally exists in several brothers and sisters, the parents being unaffected. Similar cases may be seen without a family association. There are records of many families in which jaundice has existed through three or four generations. It is transmitted alike through the male and female descendants, and not all of the children in a family are affected. The descendants of unaffected members escape. The jaundice may be noticed shortly after birth, or it may develop at any time during childhood, sometimes not until later. This is the most striking feature of the disease. The discolouration may be very slight and noticeable only in the sclerotics, or the skin may be icteric. The colour is never very intense. It varies somewhat in degree and is increased after intercurrent gastro-intestinal attacks, which are rather frequent. When once developed, the icterus never entirely disappears.

This jaundice is not obstructive; the stools are usually darker than normal and the urine contains urobilin in excess, but no bile. There is an increased production of biliary pigment. The liver is normal or slightly enlarged. The spleen is regularly, and often excessively, enlarged, and even in youth there may be attacks of biliary colic and of perisplenitis. Anæmia of a moderate grade is the rule. Both the red cells and hæmoglobin are reduced; and a few nucleated red cells may be found. Very characteristic of the disease is the increased fragility of the red cells to hæmolytic agents, especially to hypotonic salt solutions.

The growth and development of children go on uninfluenced by the

condition, and many affected persons have lived to an advanced age. There are no characteristic post-mortem findings, and the disease is uninfluenced by treatment.<sup>1</sup>

#### CATARRHAL JAUNDICE.

This is due to a catarrhal inflammation of the common bile duct with which there is usually associated a similar inflammation of the duodenum and sometimes of the stomach also. The term *gastro-duodenitis* is sometimes used synonymously with catarrhal jaundice. The jaundice in these cases is due to obstruction which is caused by swelling of the mucous membrane of the bile duct. Catarrhal jaundice is rare in infancy. I have never seen it in a child under two years old. In children from three to six years it is not uncommon, and curiously occurs much more frequently in the fall months. This suggests an infectious origin. For the most part its causes are obscure.

It occasionally complicates malarial fever. I have seen it several times with influenza, and it may occur with any of the infectious diseases. Rehn has described a form which occurred epidemically.

The symptoms of the disease are quite uniform. When primary, the onset is like an ordinary attack of indigestion, with vomiting, pain, slight fever, and a moderate amount of prostration. The vomiting in some of the cases is repeated for several days. The pain may be quite severe, and localised in the region of the duodenum. It may be associated with tenderness in this region. The bowels are usually constipated. After three or four days, icterus, which is the only diagnostic symptom, appears. It is first seen in the conjunctiva, afterward in the skin, varying in degree according to the severity of the attack, but in most cases not being very intense. It is accompanied by the regular symptoms of obstructive jaundice. The stools are gray, sometimes white; there is a marked amount of intestinal flatulence. The urine is very dark, of a yellowish-green or bronze hue, and stains the clothing. There is complete anorexia; the tongue is thickly coated with a white fur. Headache, dulness, and languor are present, and the patient feels generally wretched. The slow pulse and the itching skin are uncommon symptoms in children. The liver is usually found, upon examination, slightly enlarged, and sometimes tender on pressure. The duration of the disease is about two weeks, the general symptoms disappearing before the icterus. Recurrences and prolonged attacks are occasionally seen.

The diagnosis rarely presents any difficulty, and the prognosis is invariably good.

**Treatment.**—In the diet, fats and starches should be reduced to a low point or be entirely prohibited. Patients usually do much better

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<sup>1</sup> Tileston and Griffin, American Journal of the Medical Sciences, June, 1910.



upon a diet of rare meat, fruit, and of skimmed milk, or buttermilk. If there is very much vomiting, the milk should be largely diluted with lime-water. The amount of food given should be small, but water should be allowed freely, particularly the mineral waters. The bowels should be opened every other day by calomel, followed by a saline purgative. In most of the cases no other treatment is necessary. When the pain is severe it may be relieved by counter-irritation by mustard, turpentine, or even cantharides. The gastric symptoms should be managed as are those of ordinary acute gastritis. The restricted diet should in all cases be continued for at least a week after the jaundice has disappeared.

#### FUNCTIONAL DISORDERS OF THE LIVER.

Functional disorders of the liver are undoubtedly exceedingly common in childhood. They are as yet but little understood, and it is almost impossible to separate them from the other symptoms of intestinal indigestion with which they are associated. These are described in the chapter upon Chronic Intestinal Indigestion. Some of these symptoms depend upon a diminution in the quantity, or the impoverished quality of the biliary secretion. There are gray or white stools, flatulence, and other evidences of increased intestinal putrefaction. These probably depend upon imperfect absorption in consequence of the absence of bile. The other functional disorders of the liver relate to its effect upon the transformation of nitrogenous substances. The nature of this change, and the symptoms which result from this disturbance are as yet but imperfectly understood. It is quite probable that many of the nervous functional disorders of children—for example, attacks of migraine or of cyclic vomiting—may depend upon such a cause.

#### NEW GROWTHS.

New growths of the liver are rare in children and are usually secondary to deposits elsewhere, most frequently in the kidney. They are generally sarcomatous. Primary sarcoma of the liver has, however, been observed, and at so early an age as to make it practically certain that the condition was a congenital one. A single example of primary adenocarcinoma of the liver has fallen under my observation. This was in an infant only seven months old. In a report of this case I collected from the literature ten cases of sarcoma of various types in infants under one year.<sup>1</sup> In most of the cases there is simply a slowly increasing abdominal tumour and progressive asthenia.

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<sup>1</sup>Archives of Pædiatrics, April, 1905.

## ACUTE YELLOW ATROPHY.

This form of hepatic disease is very rare in children. Greves has reported a well-marked case in an infant of twenty months, and has collected seventeen other cases under ten years of age; the youngest was in an infant three months old. The symptoms and course of the disease are essentially the same as in adults. A condition closely allied to this is occasionally seen as a result of the administration of chloroform.

## CONGESTION OF THE LIVER.

Congestion of the liver occurs from the same causes in children as in adults. Acute congestion is not often seen. Chronic congestion is more common, and is usually secondary to general venous obstruction dependent upon congenital or acquired heart disease, atelectasis, or other pulmonary conditions, particularly chronic pleurisy, chronic interstitial pneumonia, and emphysema. Chronic congestion of the liver causes no characteristic symptoms except a moderate enlargement of the organ with some pain and tenderness. The treatment is that of the primary disease.

## ABSCESS OF THE LIVER—SUPPURATIVE HEPATITIS.

In 1890 Musser found but thirty-four recorded cases of abscess of the liver in children under thirteen years. Since that time a few additional cases have been reported. In the above collection, there have not been included cases of suppurative hepatitis occurring in the newly born.

As in adults, abscess of the liver may result from traumatism, or it may be secondary to suppurative pylephlebitis, which depends upon a focus of infection in the umbilical vein, or in some part of the abdomen from which the branches of the portal vein arise. Pylephlebitis may follow appendicitis (Bernard's case), it may follow typhoid fever directly (Asch's case), or be due to suppuration of the mesenteric glands or peritonitis following typhoid. In seven of the cases collected by Musser the disease was due to migration of roundworms from the intestine into the hepatic ducts. Menger (Texas) has reported one case following dysentery, the only one, I think, on record in this country. Very rarely great numbers of minute abscesses are found as a result of suppurative thrombosis of the jugular bulb following middle ear disease. In quite a number of cases no adequate cause can be found.

In the cases occurring in pyæmia and in those associated with pylephlebitis there are usually several abscesses; in traumatic cases generally but one. If untreated, the majority of cases prove fatal either from exhaustion or from rupture into the pleura or peritonæum. In Asch's case spontaneous cure took place by rupture into the intestine.

**Symptoms.** Occasionally abscess of the liver is latent, but in most of the cases the symptoms are marked and sufficiently characteristic to make the diagnosis a matter of no great difficulty. The most constant general symptoms are chills, which may be single, but are usually repeated, fever, which is commonly of the hectic variety and followed by sweating, prostration, vomiting, diarrhoea, and cachexia. Jaundice is present in less than half the cases, and is rarely intense. The liver is almost invariably sufficiently enlarged to be easily made out by palpation or by percussion, the enlargement in most cases is chiefly downward. Pain is quite constant, and frequently intense, but not always in the region of the liver. It may be in the epigastrium, at the umbilicus, in the lower part of the abdomen, and occasionally in the right shoulder. Tenderness over the liver is usually present. A positive diagnosis of hepatic abscess is to be made only by aspiration and the withdrawal of a fluid having the characteristics of "liver pus." Pulmonary symptoms may also exist with an abscess occupying the convexity of the right lobe. These may be cough and dyspnoea from pressure, or pleurisy from extension of the inflammation through the diaphragm, or from rupture of the abscess into the lung. The usual duration of abscess of the liver after its formation is the average of from one to six months. The prognosis is unfavourable, and the mortality is high. The pyogenic cases are more fatal. In Hunter's collection, he reported that of 100 cases 75 were fatal. In the present collection, a number of methods of treatment have been employed, but the results have not been such as to warrant the recommendation of any one method.

**Prognosis.** In the majority of cases the prognosis is unfavourable, and the mortality is high. The prognosis is more favourable in the pyogenic cases, but still remains uncertain. In the present collection, the results have not been such as to warrant the recommendation of any one method.

#### DISCUSSION

The following cases are from the collection of the University of Michigan, and are given to illustrate the various methods of treatment employed, and the results obtained. The first case is from the collection of the University of Michigan, and is given to illustrate the various methods of treatment employed, and the results obtained. The second case is from the collection of the University of Michigan, and is given to illustrate the various methods of treatment employed, and the results obtained. The third case is from the collection of the University of Michigan, and is given to illustrate the various methods of treatment employed, and the results obtained. The fourth case is from the collection of the University of Michigan, and is given to illustrate the various methods of treatment employed, and the results obtained. The fifth case is from the collection of the University of Michigan, and is given to illustrate the various methods of treatment employed, and the results obtained. The sixth case is from the collection of the University of Michigan, and is given to illustrate the various methods of treatment employed, and the results obtained. The seventh case is from the collection of the University of Michigan, and is given to illustrate the various methods of treatment employed, and the results obtained. The eighth case is from the collection of the University of Michigan, and is given to illustrate the various methods of treatment employed, and the results obtained. The ninth case is from the collection of the University of Michigan, and is given to illustrate the various methods of treatment employed, and the results obtained. The tenth case is from the collection of the University of Michigan, and is given to illustrate the various methods of treatment employed, and the results obtained.

The anatomical features of cirrhosis in early life are essentially the same as in adults. The liver is sometimes enlarged, but usually it is smaller than normal. The connective tissue may be distributed around the lobules, along the bile ducts, in irregular patches, or in striations through the organ. Associated with this there is atrophy and fatty degeneration of the liver cells. In some of the cases reported there has been also a similar increase in the connective tissue of the spleen and kidneys.

**Symptoms.**—These are very much the same as in adult life. In the beginning there are the indefinite disturbances referable to the digestive organs, and the liver may be slightly enlarged; later there is ascites, enlargement of the spleen, and dilatation of the abdominal veins. Ascites is a pretty constant symptom, and is generally marked. Slight icterus is often present, but a marked amount is rare. There may be hæmorrhages from the stomach, from the nose, or from other organs; in a few cases there is slight fever. The late symptoms are, a small liver, marked ascites with the consequent embarrassment of respiration, cachexia, and sometimes general dropsy. Diarrhœa is a much more constant symptom than in adults. Death usually takes place from exhaustion. The course of cirrhosis in children is commonly more rapid than in adults, and the progress is steadily downward.

**Treatment.**—Medicinal treatment is of avail only in cases which are syphilitic. These should be put upon anti-syphilitic remedies in full doses. The treatment in other respects is symptomatic and palliative. As largely as possible patients should be kept upon a milk diet. The ascites may require paracentesis as in adults.

#### AMYLOID DEGENERATION (*Waxy or Lardaceous Liver*).

From the experiments of Krawkow, Davidsohn, and others there seems now little doubt that amyloid degeneration can be produced by the prolonged action of the staphylococcus aureus, and probably by other organisms. Amyloid degeneration of the liver is associated with similar changes in the spleen and kidneys, and sometimes in the villi of the small intestine, and is usually seen in children after long-continued suppuration in chronic bone or joint disease, empyema, tuberculosis, or syphilis.

The liver is generally very much enlarged; in extreme cases a weight of six or seven pounds may be reached. It is of a glistening, waxy appearance, very firm and hard. With a solution of iodine, a mahogany-brown reaction is obtained. The amyloid substance is deposited between the capillaries and the hepatic cells, leading to occlusion of the vessels and atrophy of the cells from pressure.

Amyloid liver *per se* produces few symptoms. Ascites is rarely present except in cases in which the liver is very large, and jaundice does not

occur. In addition to the symptoms of the original disease in the course of which the amyloid degeneration occurs, there is the peculiar waxy cachexia which is seen in no other condition, but resembles somewhat that belonging to malignant disease. The face has the appearance of alabaster, and the skin has a singular translucency. The liver may be so large as to form a tumour, sometimes nearly filling the abdominal cavity. Not infrequently it extends to the umbilicus, and even to the crest of the ilium. The surface is smooth and hard, and the edges usually rounded. There is no localised pain or tenderness. The spleen is invariably enlarged. As a result of the associated amyloid degeneration of the kidney, there may be anasarca and albuminuria. Dropsy may occur from pressure of the large liver upon the vena cava, apart from the condition of the kidney.

Amyloid changes usually take place slowly, the whole course of the disease being marked by years, the patient dying from slow asthenia, from nephritis, or from some acute intercurrent disease. As a rule, cases go on steadily from bad to worse; but sometimes, after the disease has reached a certain point, the condition remains stationary for a long time.

The prognosis is always bad, although in a few cases improvement, and even cure, are stated to have occurred after the excision of the diseased joints upon which the amyloid degeneration depended. When due to syphilis, the usual anti-syphilitic remedies should be given.

#### FATTY LIVER.

Fatty infiltration of the liver is generally a secondary condition in early life, and causes no symptoms by which it can be positively recognised. Considerable discussion has of late arisen regarding its frequency in infants. From our records at the Babies' Hospital, Dr. Martha Wollstein has tabulated 345 consecutive autopsies in which the condition of the liver was carefully noted. The liver was fatty in 201, or 58 per cent. Of these autopsies, 63 were cases of tuberculosis, in 43 of which, or 68 per cent, the liver was fatty.

The general nutrition of the 345 infants was as follows:

|                  |      |  |
|------------------|------|--|
| Wasted           | 188: | liver fatty, 104, or 55 per cent—very fatty in 17. |
| Fairly nourished | 80:  | " " 32, " 65 " " " " 9.                            |
| Well nourished   | 77:  | " " 45, " 59 " " " " 20.                           |

These figures coincide very closely with the observations of Freeman at the New York Foundling Hospital, and indicate that fatty liver is not, as has been so often asserted, much more frequent in wasted infants than in others. The cause of this change in the liver is as yet but little understood.

The liver is moderately enlarged, smooth, with rounded edges, of a yellowish-red or a lemon-yellow colour, and can be indented with the

finger. A warm knife becomes coated with oil after cutting. Microscopically there is seen an accumulation of fat in the liver cells, usually irregularly distributed, but chiefly in the periphery of the lobule. Jaundice, ascites, and the other peculiar symptoms of hepatic disease are absent. The liver is moderately increased in size and its functions may be interfered with, but not in such a way as to be recognised by the symptoms. The treatment is that of the original disease.

#### HYDATIDS.

Echinococcus disease of the liver, while rare among adults in this country, is almost unknown in children. I have been able to find but two recorded cases in America. From twenty-two European cases collected by Pontou, it appears that unilocular cysts are especially frequent in young subjects. If the upper surface is affected, pulmonary symptoms, cough and dyspnoea, are usually present; if the under surface of the organ, there is pressure upon the portal vein, the vena cava, bile ducts, stomach, and intestines. This pressure may cause icterus, dilatation of the superficial abdominal veins, and sometimes ascites. The local signs are enlargement of the liver with a tumour, which is easily recognised in children because of the thin abdominal walls. The hydatid fremitus is usually obtained. By aspiration a clear fluid is withdrawn, showing under the microscope the presence of the hooklets, which establishes the diagnosis. Occasionally cure may take place by spontaneous rupture or suppuration of the cyst, but in most cases, when left to itself, the disease proves fatal. The treatment is surgical, and consists in aspiration or in incision, and the evacuation of the cyst.

#### BILIARY CALCULI.

Up to the age of puberty calculi are extremely rare. Of twenty cases collected by Still, eleven occurred in newly-born infants or else gave symptoms during the first month of life. The prominent symptom was intense and persistent jaundice. Nearly all died within the first month, the autopsy usually showing multiple calculi in the common duct.

The cases in older children do not differ from those in adults.

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### CHAPTER XII.

#### *DISEASES OF THE PERITONÆUM.*

INFLAMMATION of the peritonæum is not very frequent in childhood, because at this time most of the causes which are operative in later life either do not exist at all or are infrequent.

We shall consider separately acute, chronic, and tuberculous peritonitis.

#### ACUTE PERITONITIS.

Acute peritonitis may occur at any period of infancy or childhood. It may even exist in intra-uterine life. In the newly born, peritonitis is not infrequent. After this time it is exceedingly rare during infancy, only four cases, including all varieties, being met with in 726 consecutive autopsies in the New York Infant Asylum. After the fifth year the disease is relatively much more common. Of the 187 cases above referred to, 25 per cent occurred in the newly born, 21 per cent between one and five years, and 54 per cent between the fifth and the sixteenth years.

**Etiology.**—In the newly born, peritonitis is seen as one of the most frequent lesions of acute pyogenic infection. It is usually due to direct infection through the umbilical vessels. In infancy and childhood, peritonitis occurs both as a primary and secondary inflammation. The primary form is rare. It may be due to traumatism, such as falls or blows, or to surgical operations upon the abdomen; it has occurred after an injection for the cure of a congenital hydrocele. In a very small number of cases the inflammation seems to have been excited by cold or exposure, and it may follow severe burns. Cases of acute serous or suppurative peritonitis are occasionally seen which are apparently primary. I have met with two such in young children which were due to the streptococcus.

The secondary form is more common. The most frequent of all causes is appendicitis, which should always be suspected in acute peritonitis occurring without definite cause. Extension of inflammation from the viscera to the peritonæum is very much less frequent in children than in adults. I have seen it but once in autopsies in acute intestinal diseases. It is also rare in typhoid fever, being noted but twice among my collected cases. It is occasionally due to abscess of the liver, ulcer of the stomach, acute intestinal obstruction from internal strangulation, intussusception, volvulus, or congenital atresia. It may extend from inflammation of the pleura. This may be in the form of an empyema which burrows through the diaphragm, or, without burrowing, the infection may take place through the lymph channels; or it may be secondary to a general pneumococcus septicæmia. Peritonitis is infrequently due to infection through the female genital tract, especially in gonococcus vulvo-vaginitis in older girls. Extension of inflammation from the male genital organs is very rare. In one case at the New York Infant Asylum, fatal peritonitis in an infant started from a suppurative inflammation of the tunica vaginalis of unknown origin, the infection extending into the peritonæum through the inguinal canal.

Any abscess in the neighbourhood may rupture into the peritonæum and excite peritonitis. Those most frequent in children are connected with Pott's disease, perinephritis, and cellulitis of the abdominal wall.

Of the acute infectious diseases, peritonitis is most frequently seen with pneumonia, and very rarely with scarlet fever. When secondary to pneumonia, there is usually extreme pleurisy and sometimes also pericarditis and meningitis; in other words a general pneumococcus infection is present.

The bacteria most frequently associated with acute peritonitis in children are: the streptococcus, especially in the newly born; the pneumococcus in cases complicating pneumonia or empyema; and the *b. coli communis*, associated with other pyogenic bacteria, in those following intestinal perforation.

**Lesions.**—In the fibrinous form there are changes similar to those occurring in inflammation of the pleura and the other serous membranes. The peritonæum is injected and fibrin is thrown out in considerable quantity, usually accompanied by a small amount of serum. The process is usually a localised one. The peritonæum lining the abdominal wall, as well as that covering the adjacent coils of intestine and the solid viscera, is covered by patches of yellowish-gray fibrin, causing adhesions between the various viscera and often matting the intestines together. In recent cases these adhesions are soft, and easily broken down; in old cases they are quite firm, and they may result in the formation of connective-tissue bands which are the source of subsequent trouble. In other cases the serum is more abundant, usually clear, but it may be turbid or even bloody.

In the purulent form the products are serum, fibrin, and pus. When peritonitis results from perforation it is, as a rule, purulent from the outset, and the pus is foul and stinking. The amount of pus is proportionally larger than in adult cases. When the disease proves fatal in a few days there is found an extensive exudation of fibrin, with the formation of small pockets containing pus, among the coils of intestine. Occasionally there may be larger collections of pus in the peritoneal cavity. In cases which have lasted a longer time—generally those of localised inflammation—the process results in the formation of a peritoneal abscess. This consists in a collection of pus in some part of the peritoneal cavity, the situation depending upon the cause, but it is usually in one iliac fossa or in the pelvis. The abscess is shut off from the rest of the peritoneal cavity by a thick wall of fibrin. If left alone, such abscesses may open into the rectum, vagina, bladder, pelvis of the kidney, or externally, usually at the umbilicus. After the discharge of pus the cavity may contract and fill up by granulations, and the patient recover.



Inflammations of the other serous membranes, especially the pleura, are often associated with peritonitis.

**Symptoms.**—The symptoms of acute peritonitis in older children, as in adults, are usually well marked and sufficiently characteristic to enable one to recognise the disease easily; but not so in the case of infants. In them the symptoms are often obscure, and the disease may be found at autopsy when not suspected during life. The onset is nearly always abrupt, with fever and vomiting. As a rule, the temperature is high—from 103° to 105° F. Vomiting may occur only at the onset, but it often continues; the vomited matters are usually green. Older children complain of pain, which may be localised or general; and in younger ones this is indicated by crying and fretfulness. The abdomen very soon becomes swollen and tympanitic, this being one of the most constant features of the disease. The distention is generally uniform, but it may be irregular. There is tenderness on pressure, and usually marked rigidity of the abdominal walls. The pain causes the child to assume a fixed position and he cries if moved or disturbed. The posture is generally dorsal, with the thighs flexed. The bowels are in most cases constipated, but diarrhoea is by no means rare. The abdominal distention causes dyspnoea and thoracic breathing. There may be retention of urine or frequent micturition.

The general symptoms, almost from the beginning, are those of a serious disease. The pulse is small, rapid, and compressible. The prostration is great, from the very outset. The face is pinched, the mouth is drawn, and the features indicate pain. In severe cases there may be hiccough, cold extremities, clammy perspiration, and collapse. The mind is usually clear. In infants there may be convulsions. A polymorphonuclear leucocytosis is almost invariably present, but is wanting in some cases of the gravest type.

In the most severe forms of general peritonitis the course is short and intense, and the disease goes on rapidly from bad to worse until death occurs. In infants this is often on the third or fourth day. The very severe forms of general peritonitis in older children run the same rapid course. In other cases the course is slower, lasting a week or ten days. If the patient lives longer than this the case is more hopeful, because the process is more apt to be localised. The development of peritoneal abscess is indicated by the continuance of the temperature, which may assume a hectic type, and be accompanied by chills and sweating. There are the local signs of an abdominal tumour.

**Prognosis.**—Acute general peritonitis, whatever its cause, is a very serious disease in childhood. Of eighty cases of all varieties under sixteen years of age, sixty-nine per cent died. In the newly born and in infancy the disease is almost invariably fatal. In older children the outlook is not quite so hopeless, and depends upon the exciting cause.

**Treatment.**—The medical treatment of acute general peritonitis in children is extremely unsatisfactory, as the disease is almost always fatal unless it can be relieved surgically. Opium is indicated only for the relief of the single symptom, pain. It has, however, serious disadvantages in that it may mask important symptoms. Other medical treatment is symptomatic only and is to be employed in conjunction with surgical measures.

As a local application cold is usually to be preferred. It may be applied either by an ice-bag or by a Leiter's coil. If children rebel against the use of cold, heat may be substituted. Turpentine stupes may aid in relieving tympanites.

Feeding is always a difficult matter on account of the strong tendency to vomit; this is due to regurgitation from the intestine into the stomach, which in some cases is almost continuous. In such conditions I have found great benefit from washing the stomach shortly before feeding, repeating this several times each day. In this way vomiting may often be controlled and the stomach made ready for food. The diet should be peptonised milk, broth, or kumyss.

In every case of acute peritonitis, an immediate exploratory operation should be done if the child's general condition will permit. Appendicitis is often found to be the cause when least expected; and even when the peritonitis is due to some other cause operation gives the only chance for recovery. Operation is also indicated in localised inflammations with the formation of peritoneal abscesses.

#### CHRONIC (NON-TUBERCULOUS) PERITONITIS.

Peritonitis may occur in foetal life with the production of extensive adhesions, which may interfere with the development of the intestine and result in various malformations. These cases have been ascribed by Silbermann to syphilis.

Chronic peritonitis may follow the acute form, in which there are left adhesions which slowly increase owing to the production of new connective tissue. Such cases are sometimes chronic from the beginning.

The peritoneal abscesses which follow the suppurative form may run a chronic course. Chronic localised peritonitis may occur in connection with disease of any of the organs covered by the peritonæum.

**Chronic Peritonitis with Ascites.**—In most cases this is chronic from the outset and independent of the causes above mentioned. By far the most frequent form of inflammation is that due to tuberculosis, and by some writers the opinion is still held that chronic peritonitis with ascites is always tuberculous. After the observations reported by Henoeh, Vierordt, Fiedler, and others, there seems to be no longer any room for doubt.

ing the existence of a chronic non-tuberculous form of peritonitis with ascites, although it must be considered a rare disease. In its pathological and clinical aspects it is to be compared to subacute or chronic pleurisy with effusion.

*Etiology.*—Nearly all the cases thus far reported have occurred in children over six years old. The causes are for the most part obscure. It may be associated with disease of the intestines or the solid viscera of the abdomen, especially with new growths of the kidney, liver, etc.

*Lesions.*—The post-mortem observations thus far have been few. In the reported cases there has been found a large amount of greenish serum in the general peritoneal cavity, with a very moderate amount of fibrin and with adhesions, which are sometimes few and sometimes very numerous. Chronic pleurisy may be associated.

*Symptoms.*—The early symptoms are of a very indefinite character, but often nothing whatever is noticed until the swelling of the abdomen begins. The enlargement comes on rather gradually in the course of a few weeks. Pain is slight, or wanting altogether. There may be some abdominal tenderness. The abdomen is usually distended with fluid, the umbilicus protruding, and the superficial veins prominent. The enlargement is generally regular and symmetrical, and the wave of fluctuation is readily obtained. The general symptoms are very few. In some cases there is a slight evening rise of temperature of one or two degrees. There may be general weakness, loss of appetite, and moderate anæmia.

The usual course of the disease is for the fluid to remain for a time and then undergo slow absorption. In some instances there is no tendency to absorption of the fluid, the general health is gradually undermined, and the patients die from exhaustion or from some intercurrent disease. The diagnosis rests upon the presence of ascites, developing gradually without any signs or symptoms of disease in the heart, liver, or other organs. The points which distinguish it from tuberculous peritonitis are considered under that disease. The prognosis must be guarded on account of the difficulty in making a positive diagnosis from the tuberculous form.

*Treatment.*—It is important that the patient should be kept at rest, preferably confined to bed. The best results are obtained by the adoption of a general tonic plan of treatment. When there is no tendency to absorption after a thorough trial of the above measures, and especially when the patient's general health begins to suffer, the fluid should be removed by paracentesis. If it continues to accumulate after repeated tapping, laparotomy may be performed, for in some cases this has the same beneficial effect as in tuberculous peritonitis.

## TUBERCULOUS PERITONITIS.

The peritonæum is quite frequently the seat of tuberculous inflammation in early life. It occurs especially between the ages of one and five years, but is infrequent during the first year. Of 100 cases observed by Still, the largest number were seen in the second year of life. In 255 autopsies upon tuberculous patients, most of them under three years old, of which I have records, the peritonæum was involved in 8.6 per cent; but in a majority of these the peritonitis was not the most important lesion nor the cause of death. Tuberculous peritonitis is apparently much more frequent in Europe than in this country. Thus, Still states that this was the cause of death in 16.8 per cent of his tuberculous patients under twelve years of age, and in 12 per cent of the deaths from tuberculosis under two years. In 105 autopsies, for the most part upon older tuberculous children, Ashby found the peritonæum involved in 36 per cent. In 883 collected autopsies upon tuberculous children of all ages, Biedert found the peritonæum involved in 18.3 per cent. These figures do not represent the number of cases of tuberculous peritonitis, as in many of them only a few miliary tubercles were present.

It is possible for peritonitis to occur as the primary lesion of tuberculosis, the bacilli entering by way of the intestine, causing no lesion of the mucous membrane, but in the great majority of cases it is secondary to tuberculosis of the intestine, the mesenteric glands, the pleura, or to that of more distant parts, such as the lungs, the bronchial glands, etc. In a small number of cases there is a history of some local exciting cause, such as a fall or blow upon the abdomen. The bovine type of the tubercle bacillus is more frequently found in tuberculous peritonitis than in any other form of tuberculosis, possibly excepting cervical adenitis, which fact is strongly suggestive of milk as the source of infection.

Tuberculous peritonitis is usually associated with other abdominal lesions—tuberculosis of the mesenteric glands, intestinal ulceration, etc. It is very rarely acute, but usually occurs as a subacute or chronic disease.

The peritonæum may be involved as one of the lesions in acute or subacute general miliary tuberculosis. This is the most common form seen in infants. The lesions consist in a deposit of miliary tubercles, which are generally rather sparsely scattered over the peritonæum. The evidences of inflammation are very slight, or they may be absent altogether. These cases do not come under observation as cases of peritonitis, as there are no abdominal symptoms.

The principal anatomical and clinical varieties of tuberculous peritonitis are the ascitic and the fibrous forms.

**The Ascitic Form.**—This is much less frequent than the fibrous form. The peritonæum is thickly sown with miliary tubercles, both discrete

and in conglomerate masses. They are found in the omentum and the mesentery, upon the surface of the intestines and the solid viscera. The peritonæum shows in varying degrees the changes of acute or sub-acute inflammation. There is congestion, with the production of a moderate amount of fibrin and a large amount of serum. In the most acute cases the fluid is in the general peritoneal cavity. In those of longer duration it may be sacculated. The fluid is usually abundant, but not excessive. It is most commonly an olive-coloured serum, but it may be sero-purulent, or even bloody. There are commonly other lesions of tuberculosis in the body, but they are usually less marked than those of the peritonæum.

Clinically, ascitic cases usually present the symptoms of a low grade of peritonæal inflammation. The onset is gradual, with indefinite general symptoms. There is usually some fever—100° to 101.5° F. There is general weakness, prostration, and some loss of flesh, but not rapid emaciation. Vomiting is not prominent, and pain and tenderness are often absent. There may be nothing distinctive until distention of the abdomen is seen. This at first is due to intestinal gas, but later to fluid, which may accumulate in sufficient quantity to fill the general peritoneal cavity. The bowels may be constipated or there may be diarrhœa. In other cases there may be only a slowly developing ascites without any inflammatory signs, and the abdominal enlargement is practically the only symptom.

The ascitic form of tuberculous peritonitis may result fatally, death occurring from general tuberculosis or by slow exhaustion from the local disease; the duration under these conditions is usually from two to four months. At other times the fluid may gradually undergo absorption and recovery take place, or after absorption the fibrous form of inflammation may develop.

**The Fibrous Form.**—This is generally slower in its development and more chronic in its course than the ascitic form. There is a tuberculous inflammation, the products of which have undergone transformation into fibrous tissue. The most important feature of these cases is the production of extensive organised adhesions between the solid viscera and the intestines, between the intestinal coils, and between the intestines and the abdominal walls. The intestines may be compressed against the spine by bands.

These adhesions and their mechanical consequences are sometimes almost the only lesions present. In other cases there may be an accumulation of fluid, which may be sacculated or in the general peritoneal cavity. This may be serous, sero-purulent, or purulent. The omentum may be greatly thickened. There are often present in the fibrous exudate covering the intestines, in the omentum, and in the mesentery, tuberculous deposits consisting of caseous nodules or larger caseous masses,

which are frequently softened at the centre. Tuberculous deposits are found upon the peritoneal surface of the intestine, and infiltrate the intestinal walls, often leading to perforation, and sometimes to fistulous communications between adherent intestinal coils. There may also be tuberculous infiltration of the abdominal walls, accompanied by cellulitis, resulting in abscesses, which may open externally, usually in the neighbourhood of the umbilicus.

Clinically, these cases are distinguished by their slow, irregular course. They are the most chronic of all the forms. The onset is generally insidious, and fever is commonly absent. There is rarely vomiting. The bowels may be constipated or loose. For a long time the general health may remain good. The only characteristic symptom is the enlargement of the abdomen. In the early part of the disease this is chiefly from the tympanites, but later there may be some accumulation of fluid. It is rare that the inflammation remains entirely fibrinous. Ascites usually develops very slowly, but may be abundant. The adhesions of the intestines may give rise to irregularities in the outline of the abdomen. Ascites may be present for a time and then disappear spontaneously, and the general health may so improve that the patient is considered quite well. There may even be a permanent cure. In other cases, after symptoms have been absent for some time, relapses occur, and more fluid is poured out. In addition to these symptoms, others are present depending upon the mechanical effects of pressure from the contracting adhesions. There may be more or less constriction of the intestine, pressure upon the vena cava, the renal or portal veins, the thoracic duct or its branches, or upon the stomach. These conditions may give rise to dyspeptic symptoms, emaciation, œdema of the lower extremities, and albuminuria. In some cases tuberculous peritonitis is entirely latent, and it is discovered at autopsy when there have been either no abdominal symptoms during life, or only colicky pains of an indefinite character. The course of this form of peritonitis is slow and irregular; it generally lasts for from six to twelve months, although with intermissions and exacerbations it may extend over several years.

If softening and breaking down of inflammatory products take place, well-marked constitutional symptoms are usually present. These are partly from the peritonitis and partly from general tuberculosis. Fever is regularly present, the temperature usually ranging from 99° to 102° F. Sometimes it assumes a distinctly hectic type. There is progressive emaciation, anæmia, prostration, and sweating. Diarrhœa is frequent, and the intestinal discharges may at times be bloody. The abdomen is large, but not so much distended as in some of the other forms; the superficial veins are often prominent. Ascites often can not be made out by percussion, although fluid can often be found by puncture. Areas of dulness and tympanitic resonance are irregularly distributed. Nodu-

lar masses of various sizes and irregular shapes may be felt anywhere in the abdomen, but they are more frequently in the region of the umbilicus and in the right iliac fossa than elsewhere. The epigastric region may be occupied by a smooth, hard tumour—the thickened omentum—which may resemble the liver. There may be the signs of phlegmonous inflammation of the abdominal wall in the neighbourhood of the umbilicus, and even an abscess, which, after opening, may leave a fistulous communication with the peritonæum. There are usually some signs of disease in the lungs, and the pulmonary symptoms may mask those of the abdomen. The course of the disease, when softening and breaking down have taken place, is steadily progressive, the usual duration being from three to six months. Death results from the pulmonary disease, from tuberculous meningitis, from exhaustion, and occasionally it is due to accidents associated with perforation.

**Diagnosis.**—The essential symptoms of tuberculous peritonitis are an enlarged abdomen, often with evidence of fluid, wasting, colicky pain, irregularity of the bowels, nodular masses in the abdomen, and usually slight but continuous fever. In young children chronic ascites with fever usually means tuberculous peritonitis. Pouting of the navel, with induration and redness about it, is suggestive, and any chronic abscess in the neighbourhood of the umbilicus is suspicious. If the abdominal effusion is sacculated instead of diffuse, the probabilities of peritonitis are much increased. If there are added physical signs pointing to disease of the lungs or the evidence of tuberculosis elsewhere, or a positive tuberculin reaction, cutaneous or otherwise, the diagnosis is almost certain. Cirrhosis of the liver is practically unknown in infancy and early childhood. If ascites is absent, tuberculosis of the peritonæum may be suspected if there are irregular nodules or masses in various parts of the abdomen, with tenderness, emaciation, colicky pains, and, in the later stages, fever. But fever may be absent for a long time, even though local symptoms are marked. The epigastric tumour due to omental thickening may be mistaken for the liver; but it generally extends quite across the abdomen, and the upper as well as lower border can often be felt. Faecal masses may resemble tuberculous deposits, but are removed by cathartics and enemata.

The examination of the fluid drawn by aspiration is not of much assistance in diagnosis. Bacilli are very difficult to demonstrate; only by animal inoculations can the tuberculous nature of the fluid usually be proven.

**Prognosis.**—Tuberculous peritonitis is always a serious disease, but by no means a hopeless one; rather more than half of all cases recover. The younger the child the worse the outlook. It is especially bad during the first year. Many cases occurring in the second year and later recover spontaneously and entirely. The most hopeful ones are those with

ascites. But even in the fibrous form some apparently complete recoveries take place, the adhesions disappearing by absorption to a degree truly remarkable. The most unfavourable cases are those in which there is strong evidence of the breaking down of tuberculous deposits, with continuous fever and wasting.

**Treatment.**—The general treatment of tuberculous peritonitis is similar to that of tuberculosis in other parts of the body. The essentials are, rest, which should be invariably in the recumbent position, a climate mild enough to permit the patient to remain out of doors the greater part of the time, and very careful attention to feeding, with the purpose of improving the general nutrition. Under this treatment a very considerable number of patients recover, especially those who are over a year old. Such a termination is more likely if the diagnosis has been made early and if the disease is limited to the peritonæum. Drugs play but a small part in the treatment of these cases, but it is the general opinion that creosote is of some value. The carbonate may be used, or the creosote itself may be given in “pearls” or in emulsion. English authorities still attach considerable importance to the use of iodoform, which may be used, though somewhat cautiously, by inunctions (twenty grains to one ounce of olive oil), or it may be given by mouth in pill form, in doses of one-third to one-half grain three times a day. A faithful trial of these measures should be made before resorting to operation. The use of tuberculin as a therapeutic measure in these cases has not yet been tested sufficiently to enable one to speak with any positiveness of results; it demands further trial.

In cases not progressing favourably under medical treatment, the question of operation should be considered. The most favourable cases for operation are those of the ascitic variety. It may be useful also with localised or general suppuration and for the relief of intestinal obstruction occurring in the course of the disease. In the fibrous form less is to be expected from it. Operation may be done for the relief of recurring colicky pains due presumably to constriction by bands. Exploratory laparotomy is indicated in all cases of doubtful diagnosis. The existence of other foci of tuberculosis does not contraindicate operation except when these are chiefly intestinal, or when there is advanced general tuberculosis.

Aldibert has collected statistics of 52 operations, with 7 deaths and 45 recoveries. Nine patients were reported well one year after operation. It is possible that among these cases some of simple inflammation were included; of 18 cases, however, in which the diagnosis of tuberculosis was established by the microscope or inoculation experiments, all recovered, and 6 were well one year after operation. Why it is that simply opening the abdomen and draining or washing out the peritoneal cavity should have such an influence in arresting the disease, which, in a



certain proportion of instances, is certainly the case, has not yet been satisfactorily explained. In deciding the question of operation, its unfavourable results should also be borne in mind. A not uncommon consequence is injury to the intestine from the breaking up of adhesions, which may result in fæcal fistulæ. For the surgical aspect of the treatment the reader should consult works upon surgery.

#### ASCITES.

Ascites consists in an accumulation of fluid, usually clear serum, in the general peritoneal cavity. It is a symptom of the various forms of peritonitis, especially the chronic varieties described in the preceding pages. It may be due also to portal obstruction from cirrhosis of the liver, or pressure upon the portal vein by peritoneal adhesions or large lymphatic glands. It is occasionally seen in all forms of abdominal tumours. Ascites may occur in general dropsy from cardiac disease, chronic pleurisy, or interstitial pneumonia, or from any condition causing pressure upon the vena cava. It is also seen in the general dropsy of renal disease. A moderate amount of ascites is often met with in extreme anæmia or leukæmia.

Small accumulations of fluid in the peritoneal cavity are difficult of detection. Large amounts are generally easily made out. There is a uniform smooth distention of the abdomen and dilatation of the superficial veins, especially about the umbilicus. On palpation, the wave of fluctuation can be obtained by placing one hand against the abdomen upon one side and giving the opposite side a sharp tap. A similar wave may be felt when there is tympanitic distention. The two are, however, readily distinguished by having an assistant make pressure with the edge of the hand along the linea alba while the test is being made; this obstructs the wave transmitted through the abdominal wall, but does not affect that through the fluid. On percussion in the sitting posture, there is dulness below and resonance above. When the patient is recumbent, there is resonance in the median line and dulness or flatness in the lateral portion of the abdomen.

The prognosis and treatment of ascites will depend upon its cause.

**Chylous Ascites.**—This term is applied to certain cases in which the abdominal fluid contains fat. The colour may be milky-white or light brown, and the fluid, after standing, may have at its surface a thick, creamy layer. The amount of fat present has been as high as five per cent. This condition is rare in childhood. The exact pathology is as yet not well understood. In the cases which have thus far come to autopsy there has usually been found chronic peritonitis, sometimes simple, sometimes tuberculous. The lymph vessels in some of the cases have been empty, and often no obstruction of the lymph circulation

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could be discovered. The fat is believed by some to be derived from fatty degeneration of the products of chronic inflammation, but this seems hardly sufficient to explain the large amount of fat sometimes found. In some of the cases it has been due to a wound of the thoracic duct. The amount of fluid is frequently very large. The prognosis is usually bad, although Pounds has reported a case in a girl of ten years, where recovery followed laparotomy. Tuberculous peritonitis was present.

#### SUBPHRENIC ABSCESS.

In the group of cases of localised peritonitis or peritoneal abscess, must be included subphrenic abscess. This is a rare condition in childhood, and consists in an accumulation of pus just beneath the diaphragm and above the liver. Its cause may be either in the thorax or in the abdomen. It may complicate acute pneumonia, usually of the right lower lobe, by a direct extension of infection through the lymph channels. Sometimes it has been associated with phthisical cavities. In the abdomen it may be associated with disease of the liver. The accumulation of pus is sometimes very great, so that the diaphragm is crowded high into the thorax.

The symptoms and physical signs closely resemble those of empyema, and most of the cases have been operated upon with the belief that the surgeon was dealing with empyema. Meltzer has reported a case in a child of two years which followed pneumonia of the right base. At the operation only a few drops of pus were found in the pleural cavity; but there was discovered a pinhole opening in the diaphragm, from which the pus had escaped from a large subphrenic abscess. This was evacuated, and the patient recovered perfectly. Subphrenic abscesses may contain air; they are then likely to be mistaken for pneumothorax. These abscesses require incision and drainage like other forms of peritoneal abscess.

## SECTION IV.

### DISEASES OF THE RESPIRATORY SYSTEM.

#### CHAPTER I.

##### NASAL CAVITIES.

##### ACUTE RHINO-PHARYNGITIS.

(*Acute Nasal Catarrh-Coryza.*)

ALTHOUGH the symptoms of acute nasal catarrh are chiefly nasal, the principal seat of the pathological process is the rhino-pharynx.

**Etiology.**—Certain children are predisposed to attacks of acute nasal catarrh. This predisposition, as it sometimes extends to entire families, may be inherited; but more frequently it is acquired, and usually by the following mode of life: It is seen in children who get very little fresh air, because they are kept indoors unless the weather is perfect; who live in houses always overheated; whose sleeping rooms are kept carefully closed at night for fear they may take cold; who are for the same reason so overloaded with clothing that they can not engage in any active play without being thrown into a profuse perspiration. These conditions after a time result in a great sensitiveness of all the mucous membranes, but especially those of the nose and pharynx, which is much increased by residence in a damp, changeable climate. Young infants and those who are rachitic, are frequent sufferers from acute nasal catarrh. Attacks are often brought on by insufficient covering for the head, by wetting the feet, by cold and exposure, especially to street dust and the raw winds of winter and spring, accompanied by the dampness which occurs with melting snow. In susceptible children the exciting cause is often a very trivial one. A draught of cold air for a few minutes may be sufficient to excite sneezing and a nasal discharge. Atmospheric conditions are probably not the only cause of acute nasal catarrh. Micro-organisms certainly play an important part, particularly the staphylococcus aureus, pneumococcus, streptococcus, and the b. influenzae, their importance being in the order named. Although pyogenic germs are always present in the nose, they do not excite an attack of acute catarrh without the vascular changes which are produced by other causes. Acute catarrh may be sporadic or epidemic; certain forms are

contagious, being communicated by children using the same handkerchief, occupying the same bed or simply by close contact.

Acute nasal catarrh may be a symptom of measles, nasal diphtheria, or influenza, and it may accompany erysipelas of the face.

**Symptoms.**—In the mild form the changes in the mucous membrane of the nose are not great, and are usually secondary to those of the rhinopharynx, being in a large measure due to the discharge. There is redness and slight swelling. The nasal passages may be for the time quite occluded by the discharge, which is usually profuse, at first sero-mucous, and later muco-purulent. The symptoms may be very transient, sometimes passing away in a few hours, in which cases there is only a vasomotor disturbance; or they may continue and develop into a true inflammation. The discharge may excoriate the nostrils and the upper lip. At the onset there is usually sneezing, and in infants often a slight fever. In older children there is no rise of temperature except in the most severe cases. The obstruction to nasal respiration causes mouth-breathing, and the dryness and discomfort which result from it produce disturbed sleep, snuffing and difficulty in nursing, this being in severe cases almost impossible. The inflammation may extend to the lachrymal duct, involving the eyes in a mild conjunctivitis. The process often extends to the larynx and bronchi, with hoarseness and cough. There may be closure of the Eustachian tubes, causing deafness and otalgia. The chief complication for which the physician should watch is otitis.

The severe form in infants is often attended by marked constitutional symptoms; the temperature may be as high as 104° or 105° F. and sometimes fluctuates widely. The discharge soon becomes muco-purulent and is very profuse, pouring from the anterior nares and filling the pharynx. The cultures in this form most frequently show the pneumococcus and the staphylococcus aureus. Severe symptoms often continue for a week or more, the child being really seriously ill. Complications are almost always present. In most cases there is cervical adenitis and otitis. If the child is a delicate one broncho-pneumonia is apt to develop. Retro-pharyngeal abscess is not infrequently seen.

**Diagnosis.**—It is important to distinguish between a simple acute catarrh and one due to measles, influenza, nasal diphtheria, or hereditary syphilis. Measles and influenza usually cause more fever and general constitutional disturbance than does simple catarrh. Nasal diphtheria may be present when there is only a profuse discharge tinged with blood. When such a discharge persists for two or three weeks this is always to be suspected, even though the constitutional symptoms may be very slight. The only positive means of excluding diphtheria is by cultures. A persistent acute nasal catarrh in a young infant should always suggest syphilis, and the patient should be carefully watched for the development of other symptoms.

**Treatment.**—A young child suffering from acute coryza should be kept indoors in a room with an even temperature of about 70° F., the bowels freely opened, and the amount of food somewhat reduced. The only drug which seems to have much influence upon the secretion is belladonna. A good combination is that known as the "rhinitis" tablet (camphor, gr.  $\frac{1}{4}$ ; quinine, gr.  $\frac{1}{4}$ ; fluid extract of belladonna,  $\mathfrak{m}$   $\frac{1}{8}$ ); one-half a tablet may be given every hour to a child of five years.

Useful local applications are liquid albolene, oleo-stearate of zinc, or alkaline sprays, such as Seiler's solution, to clear away the secretions. If the nasal obstruction causes great interference with respiration or nursing, adrenalin diluted with a saline solution may be used with a medicine dropper.

The upper lip and nostrils should be protected by vaseline or some simple ointment. Under no circumstances should irritating or astringent injections be given. In older children inhalations of spirits of camphor may be used with some advantage.

The severe cases require more active treatment. For most of them nasal irrigation with a warm saline solution is to be advised. This should be done as in diphtheria. After cleansing the rhino-pharynx a few drops of a five-per-cent solution of argyrol may be dropped into the nostrils two or three times daily.

Prophylaxis consists in solving the perplexing question, so often put to the physician, of how to prevent children from "taking cold." This is a matter of the utmost importance, and follows what has been previously said under the head of Etiology. No amount of cod-liver oil and iron will remove this tendency to catarrh so long as bad hygienic conditions continue. Sleeping rooms should be large and well ventilated, and a window should be kept open at night, except in very severe weather or during acute attacks. The temperature of the house during the day should be from 65° to 68° F., but never above this. Children should be accustomed to go out of doors unless the weather is especially bad. So firmly rooted in the minds of the laity is the idea that acute catarrhs come from cold, that the habit of coddling delicate children is always likely to be carried to an extreme.

With every delicate and "catarrhal" child one should begin in the summer by having him live in the open air as much as possible, sleeping in a room with free ventilation, with moderate covering, and continuing the same practice into the fall and early winter. If begun gradually in this way there is little difficulty in continuing throughout the winter.

The next point to be insisted on is cold sponging immediately upon rising in the morning, especially about the chest, throat, and spine. The use of chest protectors, cotton pads, and extremely thick clothing should be prohibited. Flannel underclothing should be worn upon the chest

throughout the year, and upon the legs also in winter; the very lightest in summer, and only a medium weight in winter.

Frequently repeated attacks point to the presence of adenoid vegetations in the pharynx, and no measures are of much avail until these are removed.

#### CHRONIC NASAL CATARRH.

This term is rather loosely used to designate a chronic nasal discharge. Such a discharge is frequent both in infancy and childhood. It is a condition much neglected by the general practitioner. Patients are too often subjected to routine constitutional treatment by cod-liver oil and preparations of iodine, with the idea that such cases are "scrofulous," while local treatment is either neglected altogether, or consists only of the use of the nasal douche or syringing with a saline solution. Sometimes, when suggested by parents, local treatment is opposed by the physician in the case of young children, and a great amount of harm follows. Permanent damage to the organs of hearing, smell, speech, and respiration may result from neglecting or ignoring chronic nasal catarrh in childhood.

Chronic nasal catarrh is not to be regarded as a disease, but only as a symptom which may be due to any one of a variety of pathological conditions, each of which requires very different treatment, viz., adenoid growths of the pharynx, foreign bodies in the nose, polypi, deviation of the septum or any other congenital deformity of the nasal passages, the various forms of chronic rhinitis, and syphilis, which causes a form of rhinitis peculiar to itself.

**Adenoid Growths of the Pharynx.**—These are more fully discussed elsewhere. They are by far the most frequent cause of chronic nasal discharge in infants and young children, and should be the first suspected. Every general practitioner can easily familiarise himself with the method of digital exploration of the rhino-pharynx, by which means these growths can in most cases be easily recognised. The nasal discharge accompanying adenoid growths is due to a chronic rhino-pharyngitis. Treatment is without avail unless the growths are removed. After this is done the nasal discharge usually disappears quite promptly.

**Foreign Bodies in the Nose.**—This condition should be suspected whenever there is an abundant muco-purulent discharge limited to one nostril. Foreign bodies in the nose are quite frequent in young children. Peas, beans, beads, or shoe buttons are most frequently lodged there. The efforts at removal on the part of the child, or even of the mother, generally result in pushing the body farther into the nose. It first sets up a mechanical irritation, accompanied by pain, swelling, sneezing, and sometimes hæmorrhage. This is followed by a catarrhal inflammation, which in the course of a few days becomes purulent, and may last in-

definitely. The discharge is generally quite abundant. The symptoms point to an obstruction of one nostril, and an examination with the probe readily detects the presence of the foreign body.

In recent cases the removal of the foreign body may sometimes be accomplished by compressing the empty nostril and having the child blow his nose strongly. Often the sneezing which the foreign body excites is sufficient to remove it. Before any attempt is made to seize the body with forceps cocaine should be used, not only for the purpose of preventing pain, but in order to contract the mucous membrane so as to allow better manipulation. In many cases chloroform is necessary. In most circumstances ordinary foreign bodies can with proper forceps be extracted without difficulty. No subsequent treatment is required, except the use of some mild antiseptic to keep the nose clean for a few days, as the inflammation quickly subsides after the removal of the cause.

**Nasal Polypi.**—These are among the infrequent causes of chronic nasal discharge in childhood. They are especially rare before the seventh year, but both mucous and fibrous polypi are seen. The symptoms are those of a chronic nasal catarrh with partial or complete obstruction of one or both sides. Polypi increase in size with the occurrence of every acute coryza, and are always especially troublesome in damp weather. They may be accompanied by reflex symptoms, such as cough, sneezing, and even by attacks of asthma. There may be headache, and sometimes disturbances of smell, taste, and hearing. The symptoms are of much longer duration than in the case of obstruction from a foreign body, the discharge is not so abundant, and is not purulent. The diagnosis is made only by examining the nose with the mirror and nasal speculum.

Polypi may be removed with the forceps, but this is best accomplished by the use of the wire snare. When they have been present for a long time the accompanying chronic rhinitis may require subsequent treatment.

Deviation of the nasal septum, and other congenital deformities which cause narrowing of the nasal respiratory tract, are conditions which belong to the specialist.

#### CHRONIC RHINITIS.

**Simple Chronic Rhinitis.**—Simple chronic rhinitis existing alone is of rare occurrence in young children. In the cases so classed the symptoms are usually due to rhino-pharyngitis, which almost invariably depends upon an adenoid growth. The growth may be a small one, so that the symptoms of obstruction are slight or absent. A frequent complication is chronic enlargement of the cervical lymph glands.

The only constant symptom is an excessive nasal discharge which is usually mucous but which may be muco-purulent. It is easily removed

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by blowing the nose, if the child is old enough to be taught to do this. Children too young to clear the nose in this way, suffer from almost constant discomfort. The amount of discharge depends upon the severity of the case. It frequently causes irritation of the upper lip, which may be the seat of eczema or impetigo, especially in infants. The lip may be swollen and prominent. The condition of the external parts is aggravated by the constant disposition to pick the nose, which may be overcome by the application of a short anterior splint to each elbow.

Epistaxis sometimes occurs. The duration of the disease is indefinite; it may last for months or even for years, the symptoms in summer being insignificant, but returning every cold season. It may terminate in recovery, or, in children with flabby tissues and delicate constitution, it may be followed in later childhood by hypertrophic rhinitis.

*Treatment.*—Prophylaxis is very important. The main purpose should be to prevent attacks of acute nasal catarrh by the measures mentioned in the discussion of that disease. The general treatment should not be routine, but based upon the indications of each case. General tonic treatment is required in most cases.

Local treatment consists first in cleanliness, and, secondly, in the use of astringents in the form of powder or solution. In infants, if the discharge is abundant, the only efficient method of getting rid of it is by nasal syringing. This is attended by some risk of forcing materials into the middle ear; but if carefully done, the danger seems to me to be less than that of allowing the discharge to remain. All solutions are to be made with sterile water and used warm, either with a nasal douche or syringe. Very little force should be employed, and it may be well to have a syringe the nozzle of which does not completely fill the nostril. Either Dobell's or Seiler's solution may be employed, diluted with an equal amount of water. Ordinarily, the nose should be cleansed thoroughly twice a day, more frequently in very severe cases. Harm is often done by the overzealous use of local treatment in these conditions.

**Syphilitic Rhinitis.**—Rhinitis is seen both in early and late hereditary syphilis. Coryza, or snuffles, is one of its earliest and most constant symptoms. It usually begins between the third and sixth weeks of life, rarely after the third month. The pathological condition is a subacute catarrhal rhinitis, sometimes with the formation of superficial ulcers or mucous patches. The disease is usually attended by a profuse nasal discharge of sero-mucus or muco-pus, occasionally tinged with blood. It may continue from a few weeks to two or three months. It usually requires only constitutional treatment, and protection of the nostrils and lips by the use of the ointment of the yellow oxide of mercury diluted with four parts of vaseline. When the discharge is very abundant, any one of the cleansing solutions previously mentioned may be used as a spray.



The rhinitis of late hereditary syphilis is a very different pathological condition. There are here gummatous deposits which break down, and form ulcers of the mucous membrane and deeper tissues. There is also periostitis, with extension of the disease to the cartilages and bones of the nasal fossæ, particularly of the septum. There may be perforation of the triangular cartilage, necrosis of the vomer or nasal bones, perforation of the hard or soft palate, and at times extensive ulceration of the alæ nasi and the face. Cicatrisation may follow, causing stenosis of the nostril. These lesions in the nose are generally accompanied by deep ulceration of the pharynx and soft palate. They usually occur in children who have presented the early symptoms of hereditary syphilis, but are occasionally seen when no such history can be obtained. Such was the case in a patient recently under observation in the Babies' Hospital, who had perforation of the nasal septum and of the floor of the nasal fossæ, causing a free communication with the mouth. These are cases of true *ozæna*. The odour from the discharge is at times almost intolerable. When neglected, these cases go on from bad to worse, and may continue for years, producing unsightly deformities.

The constitutional treatment is that of hereditary syphilis in general and is discussed in the chapter upon that disease.

Locally there may be used a spray of one of the cleansing solutions already mentioned, or black wash, or a solution of bichloride of mercury, 1 to 10,000. Although improvement may take place quite promptly, the results of treatment in the late cases are often unsatisfactory, as the disease has usually progressed so far before treatment is begun that some deformity of the nose results, usually a sinking in of the bridge and flattening of the alæ, giving rise to the so-called "saddle-back" deformity.

#### EPISTAXIS.

The hæmorrhage may come from any part of the nasal fossæ, but it is generally from the anterior nares, and most frequently from the vessels of the septum. Epistaxis is a rare symptom in the hæmorrhages of the newly born, and when present indicates syphilis. It is infrequent throughout infancy, but in childhood it is quite common, occurring in boys more frequently than in girls. In the latter it is especially common about the time of puberty. Children who are kept much indoors in overheated apartments, and who have susceptible mucous membranes and flabby tissues, are particularly prone to it. The exciting cause may be a local one, like a fall or blow; it may be due to picking the nose, or to any kind of mechanical irritation; it may be associated with nasal catarrh; and it is often caused by a small ulcer upon the septum. An attack may be brought on by mental or physical excitement. It occurs as an occasional, often an early symptom, in typhoid or malarial fever,

in measles, or during severe paroxysms of pertussis. It is seen in the hæmorrhagic form of all the eruptive fevers, in certain cases of diphtheria, in hæmophilia and scorbutus, in grave anæmia, leukæmia, and in diseases of the heart and blood-vessels.

**Symptoms.**—Epistaxis is frequently preceded by a sense of fulness or pain in the head, which is relieved by the bleeding. The blood is usually from one nostril, and comes slowly by drops. The amount lost is generally small, but it may be large enough, when repeated, to produce a serious grade of anæmia even in strong children, and the hæmorrhage may prove fatal. Epistaxis may be overlooked if the blood finds its way into the pharynx and is swallowed. In most of the cases the hæmorrhage ceases spontaneously in from ten to twenty minutes, recurring at longer or shorter intervals, according to the nature of the cause. Hæmorrhage from adenoid growths of the pharynx may closely resemble that from the nose, but otherwise there can rarely be any difficulty in recognising epistaxis.

**Prognosis.**—This depends upon the cause. In the great majority of the so-called idiopathic cases epistaxis is not serious. Occurring early in the course of the infectious diseases, it does not ordinarily affect the prognosis unless it is very severe. When it occurs late, however, it is always a bad sign, and particularly so in diphtheria. It may be serious in any of the hæmorrhagic diseases or in diseases of the blood, where it is not infrequently a cause of death.

**Treatment.**—To remove the predisposition, a child should receive general tonic treatment, especially plenty of outdoor exercise, and every means should be taken, by the use of cold baths, friction, and proper food, to tone up the vascular system.

An efficient means of arresting the hæmorrhage is compression of the nose between the thumb and finger. This may be combined with the application of ice over the nose, and sometimes small pieces of ice may be introduced into the nostrils. The application of cold to the back of the neck or its use in the mouth may be of service by exciting reflex contraction of the capillary vessels. All tight clothing or bands about the neck should be loosened, and the patient kept quiet in the sitting posture. After the hæmorrhage has ceased the child should not blow his nose for some time. Adrenalin is one of the most efficient local means of checking the bleeding. Another valuable remedy is the peroxide of hydrogen, used full strength. If bleeding continues in spite of all the above measures, the anterior nares should be plugged with styptic cotton, and if this does not control it, the posterior nares should be plugged. Usually very little effect is seen from drugs given internally, although in frequently recurring hæmorrhages where no local cause can be discovered, calcium lactate should be tried; at least thirty or forty grains a day should be given to a child of five years.

In severe cases of nasal hæmorrhage recurring at short intervals without any apparent cause, ulcer of the septum should be suspected, and, if present, should be touched with chromic acid.

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## CHAPTER II.

### DISEASES OF THE LARYNX.

THE characteristic feature of laryngeal disease in infants and young children is the association of muscular spasm with all forms of inflammation. Often it is the laryngeal spasm, rather than the inflammation, which gives rise to the principal symptoms. This spasm is only one expression of the great reflex irritability of young children.

#### CATARRHAL SPASM OF THE LARYNX

(*Spasmodic Laryngitis; Spasmodic Croup; Catarrhal Croup*).

The term *catarrhal spasm*, first suggested, I think, by Goodhart, is fairly descriptive of this disease, which is characterised by a very mild degree of catarrhal inflammation associated with marked laryngeal spasm.

**Etiology.**—It is not often seen during the first six months, but is frequent from this time up to the third year. After five years it is rare. It occurs in children who are well nourished, as well as in those who are cachectic. Certain children have a predisposition to such attacks; those who have had one attack are likely to have others. The condition has many points of resemblance to spasmodic asthma which may replace it in later childhood. Heredity seems to have some influence in producing this extreme susceptibility of the air passages. Catarrhal spasm of the larynx is most frequently associated with enlarged tonsils and adenoid growths of the pharynx, sometimes with elongated uvula. The exciting cause may be exposure to cold, especially to high winds, or an attack of indigestion.

**Lesions.**—The catarrhal inflammation of the larynx affects chiefly the parts above the cords; there is congestion and dryness, and later increased secretion of mucus. To this there is added a spasm of the muscles of the larynx, especially the adductors. There is no submucous infiltration, and no tendency to œdema glottidis.

**Symptoms.**—The attack may be preceded for several hours by slight hoarseness, or by a nasal discharge. During the day the child may appear perfectly well. Usually there is heard during the evening a hollow, barking cough, at first infrequent and not severe. About midnight this is apt to increase in severity, and there is now difficulty in

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breathing. As soon as this becomes marked the child wakes, and presents the characteristic symptoms of an attack. In the mildest cases the dyspnœa is not sufficient to waken the child. In severe cases there is marked dyspnœa, especially on inspiration, and a loud stridor as the air is drawn through the narrowed opening of the glottis. This may often be heard in an adjoining room. There is seen on inspiration deep recession of the suprasternal fossa, the supraclavicular spaces, and the epigastrium; also depression of the intercostal spaces, and even of the walls of the chest. Any excitement increases the spasm and aggravates the dyspnœa. The distress may be very great; the breathing usually slow and laboured; the voice hoarse, but rarely lost; the cough stridulous, hoarse, and metallic; the pulse rapid; the temperature normal or slightly elevated, rarely over 101° F. There may be slight lividity of the fingertips and of the lips, and sometimes considerable prostration. In the course of three or four hours the attack slowly wears away and the child falls asleep. During the following day, aside from slight hoarseness and occasional cough, he is apparently well. Most of the cases are not so severe as this; there are the croupy cough, hoarseness, and general discomfort, but not marked dyspnœa. On the second night there is a repetition of the experience of the first, usually quite as severe unless affected by treatment; and on the third day a remission similar to that of the day previous. On the third night the attack, if it occurs at all, is generally a mild one. Slight hoarseness persists for several days, but otherwise the child is apparently well. Many children have such attacks every few weeks in the course of the cold season, the slightest exposure or an indiscretion in diet being sufficient to induce one.

**Prognosis.**—This is good, the disease never, I think, proving fatal, although nothing is more alarming, at least to parents, than to witness for the first time one of these severe attacks of catarrhal croup.

**Diagnosis.**—Catarrhal spasm may be confounded with laryngismus stridulus and with membranous croup. Laryngismus stridulus is relatively a rare disease, and occurs only in infancy. In it we have not simply stridulous breathing, but periods of complete cessation of respiration. These may be repeated many times during the day, and may continue for weeks, being often complicated by carpo-pedal spasm, sometimes by general convulsions.

From membranous laryngitis, catarrhal spasm is distinguished by its sudden onset, the mildness of the symptoms of inflammation, the spasmodic character of the dyspnœa, and the daily remissions. The history of previous attacks will often aid in diagnosis. In case of doubt, a positive diagnosis can often be made by allowing the child to inhale a little chloroform. This at once relieves dyspnœa due to spasm, while it has scarcely any effect upon that due to membrane.

**Treatment.**—The purpose of treatment during the attack is to produce relaxation of the laryngeal spasm. This is accomplished by the use of emetics, steam, and hot fomentations over the larynx. A favourite emetic is a tablet triturate of antimony and ipecac, gr.  $\frac{1}{16}$  each. To a child of two years, one tablet may be given every ten or fifteen minutes, until free vomiting occurs; or a teaspoonful of the syrup of ipecac and fifteen drops of the wine of antimony at the same intervals. Given at longer intervals, these remedies are useful in relaxing spasm without causing emesis. When children do not vomit after two or three doses the antimony should not be repeated, as it may produce serious depression.

Emetics have a double value if the attack is due to indigestion. If there is constipation, an enema should be given. Following the free vomiting there is generally some improvement in the symptoms, but there may be a recurrence of the spasm unless other means are employed. To prevent this, antipyrine is one of the most useful drugs. One grain may be given to a child one year old. This may be repeated every two hours if necessary. Quite as much relief as that obtained from the drugs mentioned is seen from the use of steam inhalations. For this purpose the child should be placed in a closed tent, and steam introduced from a croup kettle. This may be used in conjunction with other measures, and continued as long as necessary. Poultices or hot fomentations over the larynx are often useful. In one case in which severe spasm had recurred for eight successive nights in spite of everything that was tried, the child being in great distress from the dyspnoea, I performed intubation, which gave instant relief. Tracheotomy, however, would scarcely be advisable.

During the day following the first night attack, the child should be kept in a warm room, and it is well to continue the antimony and ipecac in doses too small to produce vomiting, e. g., gr.  $\frac{1}{16}$  each, every four hours. After 6 P.M. the doses should be doubled, and at bedtime two grains of antipyrine given. If so treated, the symptoms may not recur upon the second night, or there may be only the cough without the severe dyspnoea. The child should be confined to the house for two or three days after one of these attacks, the drugs being gradually reduced; but the antipyrine should be given at bedtime for three or four successive nights.

To prevent a repetition of the attacks and remove the tendency to them, it is most important that the child should have plenty of fresh air and cold bathing, especially cold sponging about the neck and chest. Everything which experience has shown to bring on the attack should be carefully avoided. Local causes, such as adenoid growths and hypertrophied tonsils, should receive appropriate treatment. Generally it is not necessary to exclude fresh air from the sleeping room. Although an open window on a cold, damp night may sometimes excite an attack,

plenty of fresh air regularly given tends rather to diminish the susceptibility. If the child's condition is poor, general tonic treatment is to be employed.

#### ACUTE CATARRHAL LARYNGITIS.

Acute laryngitis is not nearly so frequent as the disease just described, although it is much more severe, and may even be fatal. It occurs especially in children from one to five years of age, usually in the cold season. Predisposition to attacks is induced by the same conditions as in the case of acute rhinitis. Catarrhal laryngitis may be primary, when it is usually excited by cold or exposure,<sup>1</sup> or it may be secondary to measles, influenza, scarlet fever, or other infectious diseases. It may also be of traumatic origin, from the inhalation of steam or irritating gases.

**Lesions.**—There is a moderately intense congestion of the laryngeal mucous membrane, sometimes general and sometimes localised. This may be seen with the laryngoscope, but is not always visible after death. With the congestion there are swelling and dryness, followed by increased secretion. In the milder cases the process is limited to the mucosa. In the more severe cases it involves the submucosa also, which is congested, œdematous, and may be infiltrated with cells. The changes are especially marked in the lymphoid tissue of the subglottic region. The swelling may be sufficient to produce a very marked degree of laryngeal stenosis. In many mild and in all the severe cases there is associated catarrhal inflammation of the trachea, and often of the larger bronchi. In young children there is very little tendency to œdema glottidis.

**Symptoms.**—In the mild form, such as that which is usually seen in older children, there is hoarseness, or even loss of voice, and a laryngeal cough which is sometimes hard and teasing, always worse at night. There may be pain and soreness over the larynx. Constitutional symptoms are mild or absent, the patient not usually being sick enough to go to bed, and often rebelling even at being kept indoors. The duration of the disease is from four to ten days, with a strong tendency to relapses from slight causes.

The severe form of catarrhal laryngitis is sometimes preceded by acute coryza, or there may be mild laryngeal symptoms for a few days before the development of the more severe ones. In other cases the

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<sup>1</sup> The following case is a good illustration of a severe attack excited by cold: A rather delicate infant, eight months old, an inmate of the New York Infant Asylum, was taken out, with very slight covering, on a raw December day. In a few hours hoarseness and stridor were noticed, and the temperature was 101° F.; three hours later it was 103° F., and in spite of the usual remedies which were employed the dyspnoea had reached such a degree as to require intubation. The tube was worn only three days and the child made a prompt recovery.

disease develops rapidly and severe symptoms are present within a few hours from the onset.

When the case is fully developed the voice is metallic and hoarse, and occasionally but not usually lost. There is a hoarse, dry, barking cough, which is very distressing, and sometimes almost constant. The cough, like the voice, is stridulous, and more or less stridor is present on inspiration. There is a slight amount of constant dyspnoea, but this is scarcely noticeable unless the chest is bared. Severe dyspnoea occurs in paroxysms, usually at night. Then, we may get the signs of obstructive dyspnoea similar to those mentioned in severe attacks of catarrhal spasm. This dyspnoea is chiefly inspiratory, but in some cases it increases steadily from the beginning of the attack, and may be indistinguishable from that due to membrane. Constitutional symptoms are usually present and may be severe. The temperature ranges in most cases from 101° to 103° F., but may go to 104° or 105° F. The pulse is rapid and full and respiration is accelerated. Children sometimes complain of pain in the larynx and trachea which is increased by coughing. The symptoms are severe for two or even three days, the fever continuing with moderate prostration and paroxysms of dyspnoea, sometimes even attacks of suffocation and cyanosis. Usually after two or three days there is a gradual subsidence of the dyspnoea and the inflammatory symptoms, and the case goes on to recovery. At other times the inflammation extends downward to the large and then to the small bronchi, and finally results in broncho-pneumonia. The attack may prove fatal from laryngeal obstruction due to swelling and spasm.

**Diagnosis.**—This disease is chiefly to be distinguished from membranous laryngitis. The onset of the two diseases may be very similar, and for the first twelve hours we have no absolute means of distinguishing between them, except possibly by the use of the laryngoscope, which is often conclusive in older children but not usually so in infants. All cases, therefore, should be looked upon with a degree of apprehension. The temperature in the catarrhal is usually higher than in the membranous form. The dyspnoea is mainly paroxysmal, with daily remissions and nightly exacerbations, and is chiefly inspiratory, while that of membranous laryngitis is constant, steadily and often rapidly increasing, and is present both on inspiration and expiration. In catarrhal laryngitis the voice is not usually lost, but in the membranous form this is the rule. There can be little room for doubt when there are enlarged glands, membranous patches on the tonsils, and nasal discharge. Very often, however, all these evidences of diphtheria are wanting, the really difficult cases being those in which the process begins in the larynx. The prevalence of diphtheria and a known exposure count for something in favour of membranous laryngitis. If cultures from the pharynx show the presence of Klebs-Loeffer bacilli, diphtheria of the larynx is certain;

but no conclusions can be drawn from negative cultures. In catarrhal as well as in membranous laryngitis there may be extreme dyspnoea, cyanosis, pallor, prostration, and even death.

**Prognosis.**—This depends somewhat upon the cause of the disease and also upon the age of the patient. It is much worse when it is secondary to measles or scarlet fever. It is better in children over three years of age than in infants, also when the general condition of the child is good. The prognosis in severe catarrhal laryngitis should always be guarded, not only on its own account, but also because it is impossible at first to be certain that the case is not one of membranous laryngitis.

**Treatment.**—In all cases children affected are to be kept in bed, and the temperature of the room should be between 70° and 72° F. The diet should be light and fluid, and the bowels should be freely opened. A hot mustard foot bath should be given at the outset. Antipyrine (one grain every two hours to a child two years old) is useful if there is much spasmodic dyspnoea. For this symptom emetics are beneficial, given as in catarrhal spasm. The use of ipecac and squills in smaller doses than is required for emesis (five drops each of the syrups of ipecac and squills every two hours) may give relief, especially in the early stage, when the cough is dry, hard, and severe.

All the remedies mentioned are to be regarded as accessories to the essential treatment, which consists in the use of inhalations. The child should be placed in a tent into which steam is introduced from a croup kettle. Simple steam may be used, or turpentine, compound tincture of benzoin, lime-water, or creosote may be added. In moderately severe cases inhalations should be used for fifteen minutes every two hours; in very severe ones they should be continued the greater part of the time. Poultices or hot fomentations may be applied over the larynx. Relief is sometimes obtained by using counter-irritation by a mustard paste, but blistering should never be allowed. In my experience the local use of cold is very unsatisfactory, on account of the difficulty of applying it properly, and the objection to it on the part of young children. Stimulants may be required late in the disease, the amount of prostration being the guide to their use.

In cases of extreme dyspnoea operative interference may be needed. It is required more often in infants and young children than in those who are older. Opinions will of course differ as to when the dyspnoea has reached the danger point. One should not wait for general cyanosis. If pallor, marked prostration, and steadily increasing dyspnoea are present the case should not be allowed to go on without interference, even though one may be perfectly sure that the case is one of catarrhal inflammation only. The severity of the dyspnoea is the only guide, and more than once I have seen cases shown at autopsy to be catarrhal, which were regarded during life as undoubtedly membranous. If intubation is done,



the tube can generally be dispensed with in two or three days. Convalescence is usually rapid, but there is danger of recurring attacks during the remainder of the cold season.

#### SUBMUCOUS LARYNGITIS—ŒDEMA OF THE GLOTTIS.

These two conditions are not quite identical, although they are closely associated and may be conveniently considered together. They are both rare in early life. In true œdema of the glottis there is simply a dropical effusion into the submucous cellular tissue of the aryteno-epiglottic folds, causing them to project as large rounded swellings on either side of the superior isthmus of the larynx. They may be of sufficient size to cause serious or even fatal obstruction to respiration. With the laryngoscope they appear as pale red tumours, lying usually in contact near the base of the tongue. By the finger their presence can be quite as readily distinguished. Œdema of the glottis occurs principally in the late stages of nephritis.

In the inflammatory form of œdema, or true submucous laryngitis, there is the same sort of swelling of these structures, but in this case it is due to some active inflammation in the neighbourhood. The swelling is partly from the œdema and partly from cell infiltration. Usually all the parts surrounding the upper opening of the larynx are in a state of acute inflammation. The epiglottis may be swollen to the thickness of a finger, and easily seen by depressing the tongue.

The exciting causes may be the mechanical irritation of foreign bodies, the inhalation of steam or irritating gases, erysipelas of the neck, primary catarrhal laryngitis, or retro-pharyngeal abscess.

The symptoms in both cases consist of great inspiratory dyspnoea with attacks of suffocation, while expiration may be quite easy. In true œdema there are in addition the symptoms of the primary disease. In the inflammatory form there are the evidences of local inflammation—hoarseness, cough, pain, and difficulty in swallowing. A positive diagnosis may be made by a digital examination. The symptoms develop with great rapidity in either variety, and frequently prove fatal in a few hours.

The treatment of true œdema consists in scarification or multiple puncture, the application of ice externally, and even the swallowing of ice; in the inflammatory form, in addition, local blood-letting by leeches and, as a last resort, tracheotomy. Intubation is useless in either form.

#### CHRONIC LARYNGITIS.

The following varieties are seen: (1) A simple form usually associated with adenoid vegetations of the pharynx; (2) tuberculous; (3) syphilitic; (4) that associated with new growths.

1. **With Adenoid Vegetations of the Pharynx.**—This is not very uncommon. The larynx is kept in a state of chronic congestion by the adenoid growth, and there finally develops a slight superficial catarrhal inflammation. The symptoms may continue for many months. These cases are often treated for a long time unsuccessfully by the use of sprays, inhalations, etc., but the symptoms disappear rapidly after the removal of the adenoid growth. Similar symptoms may be associated with hypertrophic rhinitis. In this also the treatment should be directed to the primary condition.

2. **Tuberculous Laryngitis.**—This belongs to later childhood, and is rare even then. In infancy it is almost unknown. Rheindorf has reported a case in a child of thirteen months, which was regarded during life as syphilitic, but was shown by autopsy to be tuberculous. Of sixteen cases in children, reported by Rilliet and Barthez, none occurred during the first three years, and only four before the seventh year. The larynx alone may be affected, or the larynx and trachea, or the larynx, trachea, and lungs. Pulmonary tuberculosis is usually found to be present at autopsy, even though there may have been no pulmonary symptoms. Demme has reported a case of tuberculous laryngitis in a boy of four years, whose lungs were healthy, death resulting from tuberculous meningitis.

The symptoms are hoarseness, aphonia, laryngeal cough, and mucopurulent, sometimes bloody, expectoration. The sputum may contain tubercle bacilli. With the laryngoscope tuberculous deposits may be seen, but more frequently tuberculous ulceration of the mucous membrane. In children this is usually superficial, the deep destructive ulceration seen in adults being very rare.

It is to be differentiated from syphilis chiefly by the general symptoms, as the laryngoscopic appearances may be very similar. The treatment consists in keeping the ulcers as clean as possible by the use of sprays and the local application of astringent powders, like nitrate of silver and sulphate of zinc or iodoform.

3. **Syphilitic Laryngitis.**—In the early stage of syphilis the larynx is often the seat of a catarrhal inflammation, which presents nothing especially characteristic except its protracted course. The laryngitis of late hereditary syphilis is quite rare, and is liable to be overlooked because of the difficulties in the way of a thorough examination, and because the disease is usually painless.

Strauss has collected fourteen cases between the ages of three and fifteen years, and added three of his own. He states that deep-seated processes are much more rare than among adults. The parts most frequently affected are, first, the epiglottis; secondly, the aryteno-epiglottic folds; thirdly, the posterior laryngeal wall. The epiglottis was involved in twelve of fourteen cases. Usually there was only perichondritis; in

the more severe cases there was partial or complete destruction of the cartilage. In four cases papillomatous masses were seen. In five cases the process extended from the epiglottis to the epiglottic folds of one or both sides. In several instances the superior vocal cords were thickened from hyperplasia, and occasionally small tumours were formed. In only one case was there ulceration of these folds. Changes in the vocal cords and the arytenoid cartilages were rare, occurring only with extensive inflammation. The symptoms are those of chronic laryngitis: hoarseness, sometimes aphonia, and in a few cases chronic laryngeal stenosis. The diagnosis can be made only by means of the laryngoscope. In most of the cases there are present ulcerations of the palate or uvula, or scars from previous ulcers; sometimes the disease extends into the nose. Serious symptoms often result when to old syphilitic lesions there is added acute laryngitis or oedema.

In addition to the usual constitutional remedies for syphilis, and to the means ordinarily employed for the relief of chronic laryngitis, intubation may be required in these cases for the relief of laryngeal stenosis. Nowhere are its advantages over tracheotomy more striking than here. The tube must usually be worn for many months.

#### NEW GROWTHS.

New growths of the larynx are not very rare in children. Excluding the granulations which follow the use of the tracheal canula, the only one that is likely to be met with is papilloma. This may occur even in infancy. According to Rauchfuss, the majority of the cases begin during the first year. Boys are more frequently affected than girls.

The symptoms depend upon the size and location of the tumour. The earlier manifestations are usually ascribed to chronic laryngitis. There is hoarseness, sometimes loss of voice, and a paroxysmal cough; later, dyspnoea develops which often increases by paroxysms. The symptoms are slowly progressive, and it may be several months before they are sufficiently severe to attract special attention. A positive diagnosis is made only by the laryngoscope. There is seen a whitish granular tumour, sometimes pedunculated, sometimes with a broad base, attached to any part of the larynx.

The treatment of these cases belongs to the specialist. Small pedunculated growths may be removed through the mouth by means of the forceps or snare. Larger ones require tracheotomy or thyrotomy. The prognosis after removal is unfavourable, on account of the likelihood of recurrence and the danger of broncho-pneumonia. Papillomatous tumours will sometimes disappear entirely if complete rest for the larynx is secured by means of tracheotomy; but the tube must be worn for from six months to a year.

## FOREIGN BODIES IN THE LARYNX AND BRONCHI.

The aspiration of foreign substances into the larynx is not an uncommon accident in children. It usually happens from an attempt to cough, laugh, or cry while the child has something in his mouth. If the body is sharp and irregular, like a pin, the shell of a nut, or a fragment of bone, it is liable to become impacted in the larynx. If smooth, like a pea or a bead, it is usually drawn into one of the bronchi, generally the right.

When the body enters the larynx there is immediately excited a violent paroxysmal cough, with dyspnoea amounting almost to suffocation. Often the body is dislodged by this initial attack of coughing. If it becomes impacted in the larynx, it may cause sudden death by occluding the glottis; elsewhere it may excite acute laryngitis, usually of considerable severity.

The impaction of a foreign body in one of the primary bronchi, or one of the lobar divisions, is indicated by cough and a severe localised pain in the chest. There may be expectoration of blood. On auscultating the chest, there is found an absence of respiratory murmur over one lung or one lobe, according to the situation of the foreign body. Percussion gives marked dullness, the signs thus suggesting pleural effusion; or there may be increased resonance, which may even be tympanitic, owing to the emphysema which rapidly develops. If the foreign body remains impacted in one of the bronchi, it usually excites a localised inflammation, which extends to the surrounding lung and may terminate in the formation of an abscess. This may result fatally, or there may follow a prolonged illness, with hectic symptoms resembling pulmonary tuberculosis; and finally, after weeks or months, the foreign body may be expelled by an attack of coughing, and the patient recover completely.

The diagnosis of a foreign body in the larynx is made by the suddenness of the attack and the violence of the early symptoms. In older children the body may be seen with the laryngoscope, but in young children this is very difficult. The position of a metallic or solid body may be revealed by the X-ray. The prognosis is always doubtful, and depends upon the nature of the foreign body and the point at which it has been arrested. The usual cause of death either with or without operation is broncho-pneumonia.

The first thing to be tried is inversion of the patient. By this means, assisted by the cough, the foreign body is not infrequently expelled, even though it has passed below the larynx. The symptoms of laryngeal obstruction may call for immediate tracheotomy or laryngotomy, intubation not being applicable to these cases. If, after tracheotomy, the foreign body can be located in the larynx, but can not be extracted through the tracheal wound, the thyroid cartilage should

be divided in the median line. The removal of a foreign body from the bronchi or the tracheal bifurcation should be attempted only by a skilled surgeon.

### CHAPTER III.

#### DISEASES OF THE LUNGS.

##### THE PECULIARITIES OF THE LUNGS IN INFANCY AND EARLY CHILDHOOD.

**Thorax.**—The general shape of the thorax is somewhat cylindrical, the conical or dome-shape of the adult thorax not being attained until puberty. The antero-posterior and the transverse diameters are nearly equal in the newly born, but after the third year the transverse diameter is always greater, the difference increasing steadily up to adult life. On account of the shape of the chest, the lungs are situated rather more posteriorly in the infant than in the adult.

The thoracic walls are very elastic and yielding, owing to the cartilaginous condition of a large part of the framework. They are relatively thinner than in the adult, chiefly from the imperfect development of the thoracic muscles. The greater part of the thickness of the thoracic walls is due to the deposit of fat, generally abundant in well-nourished infants; but where the fat is scanty the walls are extremely thin. The capacity of the thorax is considerably encroached upon by the high position of the diaphragm, the large size of the thymus gland, and the frequent distention of the stomach and intestines.

**Respiration.**—According to Uffelmann, the rapidity of respiration during sleep at the different ages is as follows:

|  |                |
|--|----------------|
| At birth . . . . .                     | 35 per minute. |
| At the end of the first year . . . . . | 27 " "         |
| At two years . . . . .                 | 25 " "         |
| At six years . . . . .                 | 22 " "         |
| At twelve years . . . . .              | 20 " "         |

During waking hours this rate is very materially increased, and from comparatively slight disturbance it may be nearly twice as rapid.

The type of respiration in infants is diaphragmatic, and it continues to be chiefly so until after the seventh year, when the costal element gradually becomes more and more prominent. The rhythm of respiration is easily disturbed. In very young infants the regular rhythm is seen only in sleep. The lungs do not always expand equally; at certain times and in certain positions respiration may be carried on for a few moments almost entirely with one lung. For some moments it may be very superficial, and then quite deep. The length of the interval between inspiration and expiration varies much at different times. Regular

rhythmical respiration is not fully established before the end of the second year. After this time disturbances of rhythm are due chiefly to pulmonary or cerebral disease; but in infancy quite marked irregularity may have little or no significance. It is very common in all asthenic conditions.

**Structure.**—As compared with the adult, the trachea of the young child is larger; the bronchi are larger, more numerous, and occupy a greater space; the air cells are much smaller and occupy less space; and the interstitial tissue is much more abundant.

**Physical Examination.**—This requires tact and time, but yields results which are quite as satisfactory as in adults. It should be undertaken only in a room having a temperature of about 72° F., or before an open fire.

**Inspection.**—This should be made with the chest bare. There should be noted, the shape of the chest, the presence of deformities from rickets, the want of symmetry in the two sides, bulging of the intercostal spaces, whether the two lungs expand equally or not, also variations in rhythm, and the presence and extent of any recession of the soft parts or bony walls as an indication of obstructive dyspnoea.

**Palpation.**—This also should be made upon the bare skin, always with the hand well warmed. Although we can not get the fremitus of the ordinary voice, we can get that of the cry. This is usually more intense than in adults, on account of the thinness of the chest walls. We frequently get a bronchial fremitus—a vibration produced by mucus in the tubes. The position of the apex beat of the heart should be determined, it being remembered that in infancy this is normally in the mammary line, or just outside of it, and usually in the fourth intercostal space.

**Percussion.**—For the examination of the back, the child may be laid face downward upon the nurse's lap, or be seated upon her arm. For the front and the lateral regions of the chest, the child is most conveniently placed upon his side across a hard pillow. The percussion blow must be light, either with a single finger or a small percussion hammer, using a finger of the opposite hand as a pleximeter. Percussion should be made both during inspiration and expiration. The normal percussion note is somewhat tympanitic, this being due to the relatively large bronchi and the thin chest walls. This note is exaggerated in the interscapular region and beneath the clavicle, especially upon the right side. Here cracked-pot resonance may be obtained even in health. In early infancy the thymus gives dulness over the sternum as low as the third rib, sometimes even below this point, this gradually diminishing as age advances.

**Auscultation.**—This may be practised with the naked ear or with the stethoscope. A stethoscope is absolutely necessary for a thorough exam-

ination of the apices of the lungs in front and in the axillary regions. Most children are less frightened by the instrument than by the head of the physician during anterior auscultation. The physician should always auscultate the posterior part of the chest first, because he is most likely to find signs of disease there, and also because this is not so apt to frighten the infant. Every part of the chest should, however, be thoroughly auscultated, not omitting the high axillary regions. A convenient position for posterior auscultation is to have the child held over the nurse's shoulder.

The normal respiratory murmur of the infant is generally described as "puerile." In quality this has been likened to the bronchial breathing of the adult, but the resemblance is not a very close one. It is rude, rather loud, and seems very near the ear. Its peculiar character is due to the fact that the tracheal and bronchial sounds are more distinct, because not transmitted through so thick a layer of lung and chest wall. It is especially loud in the regions where the bronchi are superficial, as between the shoulder-blades and beneath the clavicles, particularly of the right side. A careful comparison of the two sides of the chest will generally enable an observer to avoid errors. The irregularity of rhythm which occurs from slight causes should be remembered, and the infant's position changed several times during auscultation, to avoid the mistake of attaching too much importance to a feeble respiratory murmur of one side.

On account of the thinness of the chest walls, there is always great difficulty in distinguishing between râles produced in the bronchi and pleuritic friction sounds. Before drawing any inference from the auscultatory signs, both lungs must be examined for several minutes, changing the child's position, and often inducing a cry or compelling a deep inspiration by other means, in order to bring out signs which otherwise may be overlooked. As auscultation is extremely difficult or impossible in a crying infant, this part of the physical examination should be made first if the child is quiet, since upon it we must chiefly depend for diagnosis. Inspection and percussion can be deferred until later.

**Peculiarities in Disease.**—There are several peculiarities connected with the respiratory organs in infancy and early childhood which must be constantly borne in mind in studying their diseases. The muscular development of the thoracic wall is feeble. The soft, yielding character of the thoracic framework causes the chest to sink in readily from atmospheric pressure whenever there is obstructive dyspnoea. On account of the small size of the air vesicles, acute congestion may interfere with their function almost as completely as does consolidation. Because of the delicate walls of the air vesicles, emphysema is readily produced in obstructive dyspnoea, but it is rarely permanent. There is a tendency to collapse, either on the part of lobules or groups of lobules, but very

rarely of an entire lobe. This is a much less important factor in the production of symptoms in acute pulmonary disease than many writers would lead us to suppose. The tendency of inflammation to spread from the large to the small bronchi is very much greater than in adults. In all forms of pulmonary disease the rapidity of respiration is much greater than in adults. Areas of consolidation often exist without appreciable changes in the percussion note, because they are superficial and are surrounded by healthy or emphysematous lung. Flatness should always suggest the presence of fluid. Disease is often overlooked, from a failure to examine the whole chest.

Probably the most common mistakes are to confound bronchial râles with friction sounds, exaggerated puerile breathing with bronchial breathing, and to overlook the existence of fluid because of the presence of bronchial breathing.

#### ACUTE CATARRHAL BRONCHITIS.

Acute catarrhal bronchitis is one of the most frequent conditions for which the physician is called upon to prescribe in children. It occurs at all ages, from early infancy up to puberty. Its frequency, however, diminishes steadily after the second year. The predisposition to acute bronchitis exists with the same constitutional conditions, and is acquired in the same manner as the predisposition to the acute catarrhal inflammations of the upper respiratory tract. (See Acute Rhino-Pharyngitis.) Bronchitis is very common in children who are suffering from rickets and malnutrition. It is much more frequent in the cold months, especially in the late winter and early spring, when there are sudden atmospheric changes and high winds. The presence of large tonsils and adenoid vegetations of the pharynx are important predisposing causes of bronchitis.

Bronchitis may be a primary or a secondary disease. The primary form is excited by cold, exposure with insufficient clothing in severe weather, wetting of the feet, or chilling of the surface in any manner. Under these conditions it may occur alone, or be associated with or preceded by acute catarrh of the nose, pharynx, or larynx. In rare cases it is caused by the inhalation of irritants. Bronchitis is an almost invariable accompaniment of measles and influenza. It is very common in pertussis, in scarlet and typhoid fevers, and diphtheria, and may occur in any acute infectious disease; it also complicates pneumonia and pleurisy. The micro-organisms associated with bronchitis are chiefly the staphylococcus aureus and the pneumococcus, often in combination; next in importance are the streptococcus and, especially in protracted cases, the influenza bacillus.

**Lesions.**—Acute catarrhal bronchitis is an inflammation of the mucous membrane of the bronchi. As a rule it is bilateral, both sides being



involved in the same degree. Localised bronchitis is secondary to some other pathological process in the lungs, usually tuberculosis, old pleuritic adhesions, or pneumonia. In acute bronchitis only the larger tubes may be affected, this usually being complicated with inflammation of the trachea (ordinary tracheo-bronchitis); or, in addition, the process may extend to the medium-sized tubes (severe bronchitis); or, in infants especially, it may extend to the smallest tubes (capillary bronchitis). In the last-mentioned form there are invariably changes in the zones of air vesicles surrounding the bronchi, and these cases are therefore more properly classed as broncho-pneumonia. In the first form the inflammation is superficial, and affects only the mucous membrane of the bronchi. In the second form it may involve the entire thickness of the bronchial wall, and in the third form it does so regularly.

The pathological changes consist in congestion and swelling of the mucous membrane, desquamation of the epithelium, and an exudation of mucus and pus-cells. At autopsy the injection of the mucous membrane is usually distinct; pus and mucus line the walls of the larger bronchi, and by pressure ooze from the cut extremities of the smaller tubes. The chief lesion of the walls of the bronchi consists in an infiltration with leucocytes. In infants dying from bronchitis, the lungs are much more frequently emphysematous than collapsed. In fact the readiness with which emphysema occurs in bronchitis is one of its distinguishing features in infancy. However, this is rarely permanent but usually subsides rapidly after the acute attack is over. There is swelling of the lymph nodes at the root of the lungs, which in most of the acute cases is slight, but in protracted cases, and after recurring attacks, may be quite marked.

**Symptoms.**—It is convenient to consider separately the symptoms in infants and in older children.

**THE BRONCHITIS OF INFANTS.**—1. *The Mild Form (Bronchitis of the Larger Tubes).*—The onset is generally gradual, and the symptoms of bronchitis may be preceded by those of catarrh of the nose, pharynx, or larynx. The change in the character of the cough, the slightly accelerated breathing, and a further rise in temperature, indicate an extension to the bronchi. The cough may be constant and severe, or very slight. There is no expectoration. The secretions are usually coughed up into the mouth or pharynx, and swallowed. This sometimes excites vomiting. At other times the mucus is coughed only into the trachea or larynx, and aspirated again into the lungs. The respirations are from forty to fifty a minute, and often accompanied by a rattling sound, due to mucus in the large bronchi or trachea. The general symptoms are not severe, and unless the infant is very young or very delicate no apprehension need be felt as to the outcome. The temperature is generally from 100° to 102° F. for two or three days, then below 100° F. A mod-

erate amount of restlessness dependent upon the severity of the cough, anorexia, and sometimes vomiting and diarrhoea, are usually present.

The physical signs in the first stage are dry, sonorous râles over the whole chest. A little later these give place to coarse mucous râles heard everywhere, but especially distinct between the scapulæ and in the infra-clavicular regions. On palpation there is usually a marked bronchial fremitus. Often there is not enough dyspnœa to cause recession of the soft parts of the chest. Unless the disease extends to the smaller bronchi and the air vesicles, the illness usually lasts about a week. Coarse râles in the chest may remain for some time after the symptoms have subsided. Relapses are exceedingly common. In a delicate or rachitic child, or in one whose surroundings are bad, one attack is likely to be followed by a succession of others, so that the child may not be really well until warm weather comes. The general health may suffer from the prolonged confinement to the house, although the patient may never have been seriously ill.

2. *The Severe Form (Bronchitis of the Smaller Tubes).*—This differs from the preceding variety mainly in the greater severity of all its symptoms. The onset may be like that just described, the severe symptoms not appearing until the patient has been sick two or three days, or they may be severe from the outset. If the latter, it is indistinguishable from broncho-pneumonia. There is cough, dyspnœa, accelerated breathing, fever, and moderate, sometimes severe, prostration. The cough is tighter, and more frequently of a short, teasing character than severe and paroxysmal. There is difficulty in nursing. Dyspnœa may be quite marked and is shown by the active dilatation of the alæ nasi and the recession of all the soft parts of the chest on inspiration. The respirations, as a rule, are from 50 to 80 a minute. The temperature for the first day or two is usually 101° or 102° F., but it may be 103° or 104° F. So high a temperature does not continue unless pneumonia develops. The prostration is in most cases more closely related to the dyspnœa and the rapidity of respiration than to the temperature. Often there is slight cyanosis.

In the beginning the chest is filled with sibilant and sonorous râles. In twelve or twenty-four hours these are wholly or in part replaced by moist râles—coarse or fine, according as they are produced in the large or medium-sized tubes. The râles are always best heard behind, but they are present all over the chest. The signs are often precisely like those of an acute asthma. This prominence of the spasmodic or asthmatic element in bronchitis is characteristic of infancy and early childhood. The respiratory murmur is feeble; the resonance on percussion is normal or slightly exaggerated. As the case progresses toward recovery, the finer râles are the first to disappear. After the acute stage has passed the loud wheezing sounds sometimes persist for two or three weeks.

At the onset of such a case it is impossible to say whether the disease will be limited to the medium-sized bronchi or will extend to the smallest bronchi and air vesicles. In young or very delicate infants, and during measles, it is very common for the disease to spread rapidly to the air vesicles. In other cases, usually in infants under six months old, there may develop attacks of respiratory failure or suffocation. These may occur in a severe case at any time, and, because of the infant's inability to empty the tubes of secretion, the dyspnoea steadily increases until the respiratory muscles are exhausted, the inspiratory force being too feeble to overcome the obstruction in the tubes. The symptoms which follow are usually ascribed to pulmonary collapse. I am, however, by no means certain that this is the correct explanation, for in autopsies made in such cases I have usually found the lungs to be the seat of acute emphysema. The clinical picture is a clear one. There is no disposition to cough or cry; the pulse is feeble; the respiration very rapid, superficial, often irregular; the skin cyanotic, and often clammy. Finally, there may be added to the others signs of carbonic-acid poisoning, dulness, apathy, and stupor. Such attacks may come on quite suddenly even in robust infants, and unless the treatment is energetic, even heroic, death often follows in a few hours, being frequently preceded by convulsions.

The usual course of the disease in infants previously in good health is that the severe symptoms continue for two or three days only, after which the temperature falls to 100° or 100.5° F., and gradually becomes normal. The constitutional symptoms usually decline with the temperature, and, except during the first thirty-six hours, they rarely give cause for anxiety. Recovery almost invariably occurs unless the disease extends to the finer bronchi.

Bronchitis is principally to be distinguished from broncho-pneumonia. The differential diagnosis is more fully considered under that disease. The most important points are that in pneumonia the temperature is higher and more prolonged, the prostration greater, the râles very often localised—being heard only behind, often over only one lung—the duration is more protracted, and all the symptoms are more severe. In nearly all cases of severe bronchitis in young children some pneumonia is present.

**THE BRONCHITIS OF OLDER CHILDREN.**—This is not nearly so serious as in infants, because the same danger does not exist of extension of the inflammation to the finer bronchi and air cells.

1. *The Mild Form.*—This is very common. The constitutional symptoms are slight, and often entirely absent after the first day. The patient is never sick enough to go to bed. The first symptoms are cough and soreness or a sense of oppression beneath the sternum. The cough is always worse at night. It is at first tight, hard, and racking; later it is loose, and in children over five years old there is usually expectoration—

first of white, frothy mucus, but after a few days it becomes more abundant, and of a yellow or yellowish-green colour, from the presence of pus. The physical signs are only coarse râles, at first dry, and later moist, but heard over both sides of the chest, in front and behind. There may be some disturbance of digestion, anorexia, constipation, or diarrhœa. The usual duration of the attack is from one to two weeks. If the patient is not kept indoors the disease may pass into a subacute form, lasting for several weeks as a protracted "winter cough," but without any other important symptoms.

Such prolonged or recurring attacks of bronchitis of a subacute form should suggest influenza or tuberculosis. A positive cutaneous tuberculin reaction renders tuberculosis probable. A careful search for bacilli in the sputum should then be made. Although they may not be found at first, repeated examinations will usually disclose them. Influenza can be determined only by sputum cultures.

2. *The Severe Form.*—The onset is abrupt, with fever, chill, pains in the back, headache, cough, and sometimes pain in the chest. There is a feeling of tightness or constriction beneath the sternum. The onset resembles that of pneumonia, except that the symptoms are less severe. The temperature for the first two or three days ranges between 100° and 103° F. It is generally highest in the first twenty-four hours. The cough resembles that of the mild form, but it is usually more severe. The expectoration is more profuse, and occasionally, in the early stage, it may be streaked with blood.

The coarse râles of the mild form are present, and in addition there are finer râles—at first dry, and later moist—heard all over the chest. Frequently, wheezing râles are heard on expiration. The duration of the attack is ordinarily from two to three weeks, the patient being sick enough to be confined to bed for three or four days only. There is frequently a cough for some time after all physical signs have disappeared. Relapses are easily excited by any indiscretion before the patient has quite recovered.

The prognosis in the primary cases is good, such almost invariably terminating in recovery, and very exceptionally passing into bronchopneumonia; but this not infrequently happens when the attack complicates measles or pertussis.

**Treatment of Bronchitis.**—To remove the predisposition to bronchitis the same means should be employed as those mentioned in Acute Rhinopharyngitis. Children with tuberculous antecedents, and those who are especially prone to pulmonary disease, should, if possible, spend the winter in a warm climate. The sleeping apartments of susceptible infants should not be too cold—never below 60° F.—but they should be well ventilated. It is important in infants and young children that mild attacks of bronchitis should not be neglected.

Every young child who has an acute catarrh of the nose, pharynx, larynx, or bronchi should be kept indoors. In every such catarrh accompanied by fever the child should be kept in bed while the fever lasts, even if the temperature does not go above 100.5° F., and is accompanied by no other constitutional symptoms. A very large number of the cases will recover promptly when no other treatment is employed than to keep the child in bed. Fresh air is indispensable. But the advantages of cold air have not yet been demonstrated. According to my experience, the wide-open windows have no place in the treatment of acute bronchitis in infants or young children in the winter and spring season. The temperature of the room should be about 70° F. The room should be well ventilated and frequently aired, the child meanwhile being removed to another room. There is a great advantage in changing the child's position in the crib and from the crib to the nurse's arms. Careful attention should be given to feeding and to the condition of the bowels. A cathartic, preferably castor oil, should be administered at the outset.

Poultices are objectionable and should not be employed. The oiled silk jacket is sometimes useful. Counter-irritation is very valuable. In infants, the best results are obtained by the frequent use of a mustard paste (see chapter on General Therapeutics). The paste may be repeated, according to indications, from two to five times a day. If properly used, it will not injure the skin.

Inhalations may, in the great majority of cases, take the place of the administration of drugs by the mouth, a very great advantage in infants. They may be used by means of the croup kettle, the child always being placed in a tent. In the early part of the disease relaxing inhalations, like simple aqueous vapour or lime-water, may be used. Later turpentine, creosote, benzoin, terebene, or eucalyptol may be added. Of these, creosote has given me the most satisfaction. Inhalations are to be used for ten or fifteen minutes from four to twelve times a day.

In infancy, expectorants may advantageously be dispensed with. For older children, antimony and ipecac may be used in the first stage. When the secretion is more abundant, creosote, turpentine, or terebene may be given. Small, frequently repeated doses usually give the best results. Opium should be given cautiously to infants. The dry, harassing cough of the early stage sometimes yields to nothing so quickly as to small doses of Dover's powder (e. g., one-tenth of a grain every two hours to a child of one year). The use of emetics to get rid of bronchial secretion is not to be advised. Stimulants are not required in most of the cases. The indications for them are the same as in pneumonia. When there is much dyspnoea of the asthmatic type, nothing works as well as adrenalin. It should be given hypodermically; the dose is two to five minims of the 1-1,000 solution. The effects are almost immediate, but often only transient.

Should attacks of suffocation and respiratory failure occur in infants, the indications are to excite respiratory movements and to get as much blood as possible to the surface and the extremities. Flagellation or spanking and the use, alternately, of hot and cold douches to the chest will sometimes induce the deep respiratory efforts desired. Other useful measures are the hot mustard bath and the mustard pack applied to the entire body. Probably the most effective of all remedies is dry cupping. The chest should be cupped front and back for five or ten minutes every few hours. Oxygen should be administered. As these symptoms are liable to recur every few hours for a day or two, a repetition of the treatment may be needed. For such patients cold air is injurious. They should be kept in a room with a temperature of 70° to 72° F.

In the non-febrile cases in older children, confinement in bed is unnecessary, but they should be kept indoors. In the early stage, with hard, dry cough, one of the best remedies is brown mixture (the *mistura glycyrrhizæ composita* of the U. S. P.). It will be found advantageous in most cases to have the formula made up with one-half the usual amount of opium. When the cough is especially hard and dry, a single inhalation may be used at bedtime. In the second stage, muriate of ammonia may be added to the brown mixture; or terebene, two or three drops upon sugar, may be given four or five times a day, and inhalations should be used several times a day.

In the more severe cases the patients should be kept in bed and counter-irritation to the chest employed. For the general discomfort, pain, headache, etc., nothing is better than phenacetine and Dover's powder (two grains of the former to one-half grain of the latter to a child of five years), repeated every three to six hours. All patients should be kept in bed as long as the temperature is above normal.

After all physical signs and constitutional symptoms have disappeared, a cough continues sometimes for weeks. Expectoration is scanty, or is wanting altogether; the cough is hard, dry, often paroxysmal, and in some cases occurs at night only. For this condition the best remedies are cod-liver oil and creosote. When these measures are not effective, a change of climate should be advised.

#### FIBRINOUS BRONCHITIS (*Bronchial Croup*).

Fibrinous bronchitis is seen in diphtheria, usually as an extension from the larynx or trachea. There is, however, another form of bronchitis attended by a fibrinous exudate, which occurs as a primary disease. This is very rare in children. Weil has, however, collected twenty cases of the primary form. The etiology is obscure. It is seen at all ages, from infancy up to puberty, and it may be either acute or chronic. From the cases thus far reported it would appear that the acute form is rela-

tively more common in children than in adults. The disease may be confined to certain branches of the bronchial tree, or it may affect all the bronchi, even to the minute subdivisions. The fibrinous membrane is found loose in the tubes or adherent. There are generally associated other pulmonary changes, such as emphysema, atelectasis or bronchopneumonia.

The acute form somewhat resembles ordinary catarrhal bronchitis. The diagnostic features are, the severity of the dyspnoea and the expectoration of tube casts from the larger bronchi, or elongated cylinders from the smaller ones, the former resembling macaroni, the latter, vermicelli. The expectorated masses are often in balls or plugs, and their peculiar character is not recognised until they are placed in water. The casts are dissolved by alkalies, especially by lime-water. After the expulsion of a large cast, improvement in all the symptoms occurs. They, however, return as the exudate reappears. The ordinary duration of acute cases is from one to three weeks.

In the chronic form there are no constitutional symptoms, but only dyspnoea and cough, often recurring in paroxysms, with the expectoration of fibrinous casts. The patient may have these attacks at intervals of a few days or weeks, extending over a period of months, or even years. There are no characteristic physical signs. The diagnosis rests upon the peculiar character of the expectoration. The prognosis in acute cases is unfavourable, the mortality being 75 per cent (Weil). Chronic cases are not dangerous to life.

**Treatment.**—This is quite unsatisfactory. To loosen the membrane and facilitate its expulsion, the most efficient means are inhalations of the vapour of lime-water and the internal administration of pilocarpine. Occasionally emetics are of value. Improvement in some of the chronic cases has resulted from the use of iodide of potassium.

#### CHRONIC BRONCHITIS.

Chronic bronchitis is not a very common disease in children, particularly in young children, one reason being that chronic emphysema, so frequently an associated condition in adults, is rather rare in early life. Chronic bronchitis always accompanies chronic pulmonary tuberculosis and chronic interstitial pneumonia, with or without the occurrence of bronchiectasis. It is seen in chronic cardiac disease, especially with lesions of the mitral valve. It may occur as a late symptom of hereditary syphilis. Excluding the varieties mentioned, it usually follows attacks of acute bronchitis, the process becoming chronic because of the patient's constitutional condition or his unhygienic surroundings. The acute attack may be primary, but it often follows measles and whooping-cough. Rickets, general malnutrition, and the lymphatic diathesis are the con-

stitutional conditions in which acute bronchitis is most likely to pass into the chronic form. Deformities of the chest, the result either of rickets or of Pott's disease, are occasionally a cause.

**Symptoms.**—The only constant symptom is cough, which is persistent, obstinate, and nearly always worse at night or early in the morning. It often occurs in paroxysms strongly suggestive of pertussis. Expectoration is not generally abundant, but in older children it is usually present, and in a few cases it is profuse. A copious morning expectoration of foetid pus or muco-pus indicates bronchiectasis. There is no fever, little or no dyspnoea, and although the patients are thin, they are not emaciated, and in many cases the general health is not much affected. There may be coarse mucous râles, or no physical signs whatever. The duration of the disease is indefinite, depending upon the cause. All these patients are better in summer than in winter, and suffer frequently from exacerbations of acute or subacute bronchitis.

The diagnosis is to be made mainly from pertussis and tuberculosis. From mild attacks of pertussis the diagnosis may be impossible except by the course of the disease. Tuberculosis may be suspected if the thermometer shows regularly a slight evening rise of temperature, if there is much anæmia, and steady loss of flesh. It may, however, be present without any of these symptoms. A positive cutaneous reaction is suggestive, but a certain diagnosis can be made only by the discovery of tubercle bacilli in the sputum.

**Treatment.**—The first indication is to treat the primary disease. In cardiac cases digitalis is the best remedy, and all sedatives are to be avoided. Attention should be directed to the general condition—rickets and malnutrition each receiving its appropriate treatment. In most cases a general tonic plan of treatment is best, particularly the continuous use of cod-liver oil. In many cases a change of climate is the only thing which is really curative. For the relief of cough, opiates are to be avoided as much as possible. The main reliance should be upon potassium iodide, creosote, and terebene, the last two being given both by mouth and by inhalation.

#### REFLEX COUGH—NERVOUS COUGH.

Strictly speaking, all cough is reflex and of nervous origin. The term "reflex cough" is, however, commonly used to denote that which occurs without any evidence of disease in the larynx, trachea, bronchi, lungs, or pleura. On account of the close connection through the vagus and its branches between the mouth, ear, throat, stomach, and thoracic organs, it is possible for cough to be produced by many forms of irritation in these organs or cavities. Clinically, the following varieties of nervous cough are observed:



1. That dependent upon rhino-pharyngeal irritation. This is the most frequent form, and is sometimes caused by an elongated uvula, but is usually due to adenoid growths of the pharynx, though enlargement of all the lymphoid tissues of the neighbourhood no doubt have a part. The cough is generally excited by an accumulation of mucus in the posterior pharynx, and is dry, tickling, or hemming in character. It occurs chiefly at night, and in some patients only then; it may begin soon after the child falls asleep and continue the greater part of the night, often for months, especially in the cold season. Formerly, such coughs were often attributed to disorders of digestion, to dentition, to otitis, etc.

2. Cardiac cough. This is usually associated with mitral disease, and is due to pulmonary congestion. The cough may be dry and hard, but when the congestion is severe there may be frothy and blood-streaked expectoration.

3. A variety which occurs usually about the time of puberty, and is often associated with anæmia, chorea, or other nervous conditions. It is a short, hacking, or teasing cough, sometimes very distressing, and it seems to be a manifestation of extreme nervous irritability.

4. A periodical night cough, which is generally ascribed to irritation of the vagus or its branches by enlarged, sometimes caseous, lymph nodes of the tracheo-bronchial group. This often occurs in severe paroxysms, the character of which is very much like pertussis. The attacks are apt to come on about the middle of the night and last for several hours. Vomiting is rare. The cough may recur regularly every night for months. On account of the loss of sleep the patient's general health may be considerably undermined.

5. A very similar cough may occur in connection with abscesses in the posterior mediastinum, due to Pott's disease.

**Symptoms and Diagnosis.**—These cases are not common in infants, but are quite frequent in older children. In nearly all the varieties the cough is worse at night, and in many it may be confined to that time. The influence of habit is often seen, the attacks coming on regularly at certain periods. The general health may not be affected, except from the disturbance of sleep. The diagnosis between the different forms is often very difficult. The precise cause in a given case is discovered only by a careful examination of the ear, nose, pharynx, heart, stomach, and lungs, and by a consideration of the patient's general condition. The symptoms by which a diagnosis of enlarged or tuberculous bronchial glands is made are discussed in another chapter. Symptoms in some respects similar to these may exist with abscesses from Pott's disease.

**Treatment.**—Opium and expectorants are not indicated, and inhalations are of little value. The only successful treatment is that which is

directed to the cause of the disease. If no cause can be found, and the cough appears to be of purely nervous origin, the best results follow the use of the bromides or the administration of antipyrine at bedtime.

#### ASTHMA.

Asthma may be defined as a vaso-motor neurosis of the respiratory tract. It is characterised by attacks of severe spasmodic dyspnoea, which may be preceded, accompanied, or followed by a bronchitis of greater or less severity. In infancy, the association of asthma with bronchitis is a very close one, and the cases present quite a different clinical picture from the disease as seen in older children, which differs in no essential points from the asthma of adults.

Writers differ very much in their statements regarding the frequency of asthma in early life, mainly because of a want of agreement in regard to what shall be included under this term. The asthmatic attacks of infants are considered by some as a stage of bronchitis, by others as distinct from that disease. Typical attacks resembling those of adult life are rare in children, and extremely so before the seventh year. However, of 225 cases of asthma reported by Hyde Salter, the disease began before the tenth year in nearly one-third the number.

**Etiology.**—The general or constitutional causes are the same in children as in adults. Asthma is often hereditary. It occurs especially in children whose antecedents have suffered from gout or from various neuroses. It often occurs in children who in infancy have suffered from eczema. The local cause may be any form of irritation in the nose or pharynx—hypertrophic rhinitis, adenoid growths of the pharynx, hypertrophied tonsils, or elongated uvula—or in the bronchial mucous membrane, as a result of previous attacks of acute bronchitis. It is probable that it may also be caused by the irritation of enlarged bronchial glands. In susceptible persons a paroxysm may be excited by high winds, by cold and damp air, indigestion, constipation, or the inhalation of various irritating substances, such as dust, the pollen of certain plants, also from contact with horses, cats, and other animals. First attacks of asthma in children are apt to follow bronchitis.

**Symptoms.**—Four quite distinct clinical types of asthma are seen in children: (1) Cases which in their onset simulate attacks of bronchitis. (2) Those in which asthmatic symptoms follow an attack of bronchitis, continuing for weeks or months, but not necessarily recurring. (3) Hay fever, or the periodical form which occurs every summer. (4) That which resembles the ordinary adult asthma, with the nervous element predominating. The prominence of the catarrhal symptoms is characteristic of all forms of asthma in children, the first two varieties mentioned being peculiar to early life.

*Attacks Resembling Acute Bronchitis.*—These cases are rare, but may be seen even in infants. The onset is sudden, with moderate fever, incessant cough, severe dyspnoea, and sometimes symptoms of suffocation—cyanosis, prostration, and cold extremities. The chest is filled with sonorous, sibilant, and soon with subcrepitant râles. Instead of running the usual course of bronchitis of the finer tubes, the symptoms may pass away very rapidly, and in forty-eight, sometimes in twenty-four, hours the patient may be quite well. It is only by the course of the disease and by recurring attacks that their true nature can be recognised. In infants this form of asthma may be fatal.

*Cases following Attacks of Bronchitis—Catarrhal Asthma.*—This form is not uncommon, though it is frequently designated by some other term than asthma—sometimes as spasmodic bronchitis, or catarrhal spasm of the bronchi. The symptoms are, however, indistinguishable from asthma, and they evidently belong in the same category. This form is usually seen in infants, being rare after the third year. Many of the patients are rachitic; others have large tonsils, or adenoid growths of the pharynx; while in still others there is every reason to suspect the presence of large bronchial glands. Usually there is nothing peculiar about the antecedent bronchitis; in most cases it is not especially severe, and is limited to the larger tubes. The febrile symptoms subside in a few days, but the cough continues, as do also the dyspnoea and wheezing. When the symptoms are fairly established they are very uniform and characteristic. The respiration is accelerated, usually to 50 or 60, sometimes to 70 or 80, a minute. The temperature from time to time may be very slightly elevated, or it may remain normal. The respiration is noisy, laboured, and accompanied by distinct wheezing.

On auscultation, there is prolonged expiration accompanied by loud, wheezing and sonorous, or sibilant râles, and occasionally coarse moist râles are heard. In cases which have lasted some time a moderate amount of emphysema can be inferred from the prominence of the infra-clavicular regions, and exaggerated resonance over the chest in front and the depression of the bases posteriorly.

These symptoms and signs may continue for three or four weeks only, and gradually wear off, or they may last as many months—if they begin in the winter or spring, often continuing until the middle of the summer. While they are constantly present, they vary in intensity from time to time, being usually much worse at night. The symptoms are always increased by exposure to a cold, damp atmosphere, by any fresh accession of bronchitis, and often by trivial digestive disturbances. The usual duration of the cases I have seen has been from two to six weeks. The cough is not usually severe, and expectoration in most cases is absent. The general health is often but little affected. With recovery from the

asthmatic symptoms the emphysema usually disappears gradually, although I have seen one severe case in which it persisted.

What proportion of these children afterward develop ordinary asthma, I am unable from personal experience to say. Some undoubtedly do, but in others which I have been able to follow, recovery has seemed to be permanent. This would appear more likely in those cases closely associated with rickets, or with other causes which disappear spontaneously with time or as a result of treatment.

*Hay Fever.*—This is very rare before the seventh, and but few well-marked cases are seen before the tenth year. In its clinical aspects it does not differ essentially from the disease as seen in adults, except possibly by the greater prominence of the bronchial catarrh.

*Ordinary Attacks of the Adult Type.*—These usually occur at intervals of a few weeks or months, depending upon the nature of the exciting cause. The beginning is usually at night, with dyspnoea, a short, dry cough, and loud, wheezing respiration. Deep recession of the soft parts of the chest is seen, as in laryngeal stenosis. There is prolonged expiration, accompanied by loud, sonorous, sibilant and wheezing râles, and the vesicular murmur is very feeble. Later, moist râles may be heard. After many attacks emphysema is present. This occurs more rapidly than in adults, and may be extreme, giving rise in marked cases to serious thoracic deformity. On account of the loss of sleep and interference with nutrition, the general health may become seriously impaired.

*Diagnosis.*—Typical attacks of asthma are easily recognised. Some of the catarrhal forms seen in infancy, however, present some difficulty, and a positive diagnosis may be impossible except by the progress of the case. The blood picture in asthma is characteristic and of much value in diagnosis. The important thing is the presence of a large number of eosinophile cells. They may form as high as 15 to 20 per cent of the leucocytes. In a series of cases examined in my clinic by Wile, the average was 10.7 per cent; the highest being 26 per cent. The eosinophilia is greatest at the height of the attack. The blood examination serves to differentiate asthma from simple bronchitis and from tuberculosis. The existence of marked eosinophilia definitely establishes the asthmatic character of some of these attacks in infancy.

*Prognosis.*—This is best in the cases of catarrhal asthma in infants, and in older patients when it depends upon some local cause which can be removed, as when the disease is due to reflex nasal or pharyngeal irritation. In the majority of other cases, asthma is likely to become chronic unless the child is removed to some climate in which the attacks do not occur. The younger the child, the shorter the duration of the disease, and the less marked the hereditary tendency, the better the prognosis.

**Treatment.**—The nose and the rhino-pharynx should be carefully examined in every case of asthma, and any pathological condition there present should receive attention as the first step in the treatment. Special importance, in children, should be attached to the removal of adenoid growths of the pharynx. I must admit, however, to have seen very few cases of asthma cured or even greatly improved by these means. During attacks, the best means of relieving the symptoms is the inhalation of fumes of nitre paper or stramonium leaves. Most of the proprietary remedies (Papier de FrunEAU, Himrod's cure, and Kidder's pastilles) contain these ingredients. The sleeping room may be filled with the fumes of these substances, or the child may be placed in a tent into which the fumes are introduced. Emetics may be employed when the attack is brought on by indigestion. To prevent the recurrence of night attacks, nothing in my experience has been so valuable as a full dose of antipyrine at bedtime—four grains at five years and six grains at ten years. Between the attacks the main reliance should be upon the syrup of hydriodic acid (for a child of five years the dose is  $\mathfrak{m}$  v to  $\mathfrak{m}$  x, t.i.d.) and potassium iodide (gr. ii to gr. iv, t.i.d.), which are to be given for a long time. Tonics are to be used in nearly all cases. Those especially valuable in asthmatic patients are cinchonidia (gr. ii, t.i.d.) and arsenic (gr.  $\frac{1}{100}$ , t.i.d.). They may be advantageously combined.

In the severe acute attacks nothing gives so much immediate relief as the use of adrenalin hypodermically—dose  $\mathfrak{m}$  v to a child of three years.

In the cases of catarrhal asthma following bronchitis, expectorants and ordinary cough remedies are useless. Cod-liver oil and the iodide of potassium are valuable in some of the cases. Others are greatly relieved by the regular use of creosote inhalations several times a day, with a nightly dose of antipyrine. The fumes of nitre and stramonium often afford no relief, and sometimes the cases are made distinctly worse by them. The best of all measures is to send the child at once to a warm, dry climate.

For all children who have had repeated attacks, whether in the form of hay fever or for those whose asthma is chiefly in the winter and spring and excited by attacks of bronchitis, the most important thing is removal to a place where they do not have the disease, and a residence there long enough to break up the tendency to recurrence. This will usually require several years. The region best suited to most asthmatics is one which is high, dry, and moderately warm. Some do exceedingly well at the seashore; others much better in the mountains. Patients often suffer less in cities than in the country. If taken early, asthma in children is frequently curable by these means; if neglected, the disease is almost sure to continue until adult life.

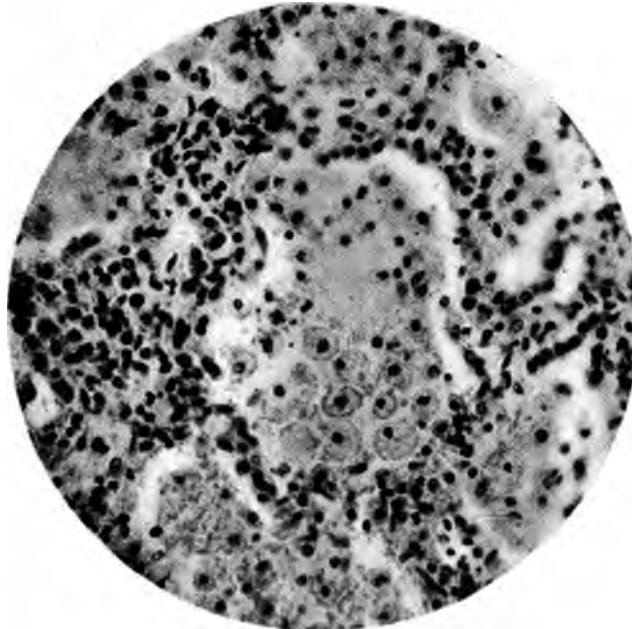
## CHAPTER IV.

*DISEASES OF THE LUNGS.—(Continued.)*

## PNEUMONIA.

In early life the lungs are more frequently the seat of organic disease than any other organs in the body. Pneumonia is very common as a primary disease, and ranks first as a complication of the various forms of acute infectious disease of children. It is one of the large factors in the mortality of infancy and childhood.

Cases of acute pneumonia are divided, from an anatomical point of view, into two principal groups: (1) Broncho-pneumonia, also known as catarrhal and as lobular pneumonia. (2) Lobar pneumonia, also known



**FIG. 70.—BRONCHO-PNEUMONIA.** The picture shows at its centre one entire air vesicle, and at its margin parts of four or five other vesicles; they are filled with large epithelial cells having small nuclei. There are also seen leucocytes with intensely black nuclei and narrow protoplasm. Between the cells is a finely granular material, which is the exudation fluid coagulated during the hardening process. The alveolar septa are somewhat infiltrated.—From Karg and Schmorl.

as croupous and as fibrinous pneumonia. These differ little from each other in etiology, but considerably in the products of inflammation, the distribution of the disease in the lung, and somewhat as to the parts involved and the nature of the changes in them.

In broncho-pneumonia the large bronchi are the seat of a superficial

inflammation, while in those of small size the entire bronchial wall is affected; the exudation into the air vesicles is mainly cellular, being made up of epithelial cells, leucocytes, and red blood-cells (Fig. 70), fibrin being either absent, or present only in small amount. In many cases there are marked changes both in the alveolar septa and in the interstitial tissue of the lung; resolution is often imperfect, and there is a strong tendency for the inflammation to pass into a chronic form, involving the connective-tissue framework of the lung. The lesion is widely and often irregularly distributed, usually being most marked in the vicinity of the small bronchi from which the inflammation spreads, and in the most superficial lobules of the lung.

In lobar pneumonia, bronchitis, when present, is usually superficial, the walls of the bronchi being very slightly or not at all affected; the



FIG. 71.—LOBAR PNEUMONIA. In the air vesicle shown in the picture there is a firm, close network of fibrin, in the meshes of which are leucocytes. At the lower part the exudation has contracted away from the wall in consequence of the process of hardening.—From Karg and Schmorl.

same is true of the alveolar septa. The principal product of the inflammation is fibrin (Fig. 71), which fills the alveoli and the terminal bronchi, the cells being relatively few and chiefly leucocytes. The process is usually sharply circumscribed, involving an entire lobe or a part of a lobe. In most cases it clears up rapidly and completely, there being but little tendency to involve the framework of the lung in a chronic process.

While in typical cases the two forms of inflammation are quite distinct, there are seen many intermediate forms which partake of the characters of both, and one may be in doubt, even after a microscopical examination, in which group to place a case. It not infrequently happens that both varieties of pneumonia are present in different parts of the same lung or in both lungs at the same time. These mixed forms are especially frequent during the second and third years; but during the first year, and after the third, the types are usually well marked.

The following table shows the relative frequency of lobar and broncho-pneumonia in three hundred and seventy cases,<sup>1</sup> nearly all taken from one institution (New York Infant Asylum). There are included all the cases of acute primary pneumonia occurring during a period of seven years:

|                   |                    |           |                  |           |
|-------------------|--------------------|-----------|------------------|-----------|
| Under six months, | broncho-pneumonia, | 73 cases; | lobar pneumonia, | 11 cases. |
| Six to twelve "   | "                  | 96 "      | "                | 29 "      |
| Second year,      | "                  | 73 "      | "                | 40 "      |
| Third "           | "                  | 19 "      | "                | 23 "      |
| Fourth "          | "                  | 0 "       | "                | 6 "       |
| Totals,           | "                  | 261 "     | "                | 109 "     |

Thus it will be seen that, of the cases of acute pneumonia occurring during the first two years, twenty-five per cent were lobar and seventy-five per cent were broncho-pneumonia.

When we come to a consideration of the micro-organisms with which the different forms of pneumonia are associated, we find that they do not correspond to the anatomical varieties. Lobar pneumonia is regularly associated with the presence of the pneumococcus, but in a large number of cases other organisms are also found. In broncho-pneumonia there is almost always a mixed infection. In the primary cases the pneumococcus is usually the predominant organism, but it is commonly associated with the staphylococcus aureus. In the secondary cases, especially when pneumonia follows measles or scarlet fever, the streptococcus is usually present, such cases being generally of a severe type. In the pneumonia of diphtheria, besides the streptococcus the diphtheria bacillus is frequently found. In winter the bacillus of influenza may be the only organism present, but it is usually associated with the pneumococcus. The organisms mentioned are found in all possible combinations, sometimes one and sometimes another predominating. With any of them the bacillus of diphtheria or that of tuberculosis may be found. Some idea of the nature of the infection in broncho-pneumonia may be gained from the following table—the sputum cultures representing the pneu-

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<sup>1</sup> The division was here made according to the predominant clinical or pathological features. Most of the doubtful cases were classed as broncho-pneumonia.



monias of one winter and spring in the Babies' Hospital, and the post-mortem cultures from those of two seasons <sup>1</sup> in the same institution :

|  | Sputum cultures from 124 cases of pneumonia. | Post-mortem cultures from the lungs in 59 cases of pneumonia. |
|--|--|---|
| <i>Staphylococcus aureus</i> . . . . . | 116  | 36  |
| <i>Pneumococcus</i> . . . . .          | 94   | 26  |
| <i>Streptococcus</i> . . . . .         | 63   | 17  |
| <i>Bacillus influenzae</i> . . . . .   | 47   | 19  |

Why the same exciting cause in one case produces broncho-pneumonia and in another lobar pneumonia may be in part owing to the difference in the structure of the lung at the different ages, especially the relatively large size of the bronchi in infancy. Again, in very young and in feeble children, the process tends to become diffuse and the products are chiefly cellular; in those who are older and more vigorous it is likely to be circumscribed, with fibrin as its chief product; in the intermediate ages and intermediate conditions the types are often mingled.

The immediate source of infection of the lungs is the mouth or the rhino-pharynx. All the forms of bacteria found in pneumonia may be found in these cavities, some of them constantly, others only at certain times, especially during an attack of any of the acute infectious diseases. Provided the other conditions are favourable, pneumonia may be excited by direct contagion. This plays a small part in inducing primary pneumonia; there seems, however, to be little doubt that the secondary forms, especially the pneumonia complicating measles, diphtheria and influenza, are not infrequently communicated in this way.

The different forms of pneumonia which will be considered are: (1) Acute broncho-pneumonia; (2) acute lobar pneumonia; (3) acute pleuro-pneumonia; (4) hypostatic pneumonia; (5) chronic broncho-pneumonia.

Tuberculous broncho-pneumonia will be discussed in the chapter devoted to Tuberculosis.

#### ACUTE BRONCHO-PNEUMONIA.

(*Catarrhal Pneumonia; Lobular Pneumonia; Capillary Bronchitis.*)

This is essentially the pneumonia of infancy. Under two years, the great majority of the cases of primary pneumonia are of this variety, and throughout childhood nearly all the cases of secondary pneumonia. The term broncho-pneumonia describes a lesion rather than a disease, several quite distinct forms of infection being included under this head. Its mortality is high, because of the tender age of the patients in which the

<sup>1</sup> See Archives of Internal Medicine, v, 449; and Journal American Medical Association, lv, 1241.

primary cases occur, and also because when secondary it complicates the most severe forms of the acute infectious diseases of children.

**Etiology.**—The 426 cases of broncho-pneumonia of which I have notes occurred as follows:

|                             |                            |
|-----------------------------|----------------------------|
| During the first year ..... | 224 cases, or 53 per cent. |
| “ “ second year .....       | 142 “ “ 33 “ “             |
| “ “ third “ .....           | 46 “ “ 11 “ “              |
| “ “ fourth “ .....          | 10 “ “ 2 “ “               |
| “ “ fifth “ .....           | 4 “ “ 1 “ “                |
|                             | <hr/>                      |
|                             | 426          100           |

After four years broncho-pneumonia is infrequent as a primary disease, although it is seen throughout childhood as a complication of the infectious diseases.

Of the cases referred to, 38 per cent occurred during the winter months, 31 per cent during the spring, 13 per cent during the summer, and 18 per cent during the autumn. While, therefore, nearly 70 per cent of the cases occurred in the cold months, broncho-pneumonia is seen throughout the year.

Broncho-pneumonia affects all classes, but is most frequent in children having poor hygienic surroundings, especially in inmates of institutions, and in those previously debilitated by constitutional or local disease. In 246 consecutive cases of primary pneumonia, 110 were in good condition prior to the attack, and 126 were delicate, rachitic, or syphilitic.

The following table gives a good idea of the conditions with which acute broncho-pneumonia is most frequently seen; 443 cases were classed as follows:

|   |       |
|---|-------|
| Primary <sup>1</sup> .....                      | 164   |
| Secondary to bronchitis of the large tubes..... | 41    |
| Complicating measles.....                       | 89    |
| “     pertussis.....                            | 66    |
| “     diphtheria.....                           | 47    |
| “     acute ileo-colitis.....                   | 19    |
| “     scarlet fever.....                        | 7     |
| “     influenza.....                            | 6     |
| “     varicella.....                            | 2     |
| “     erysipelas.....                           | 2     |
|   | <hr/> |
|   | 443   |

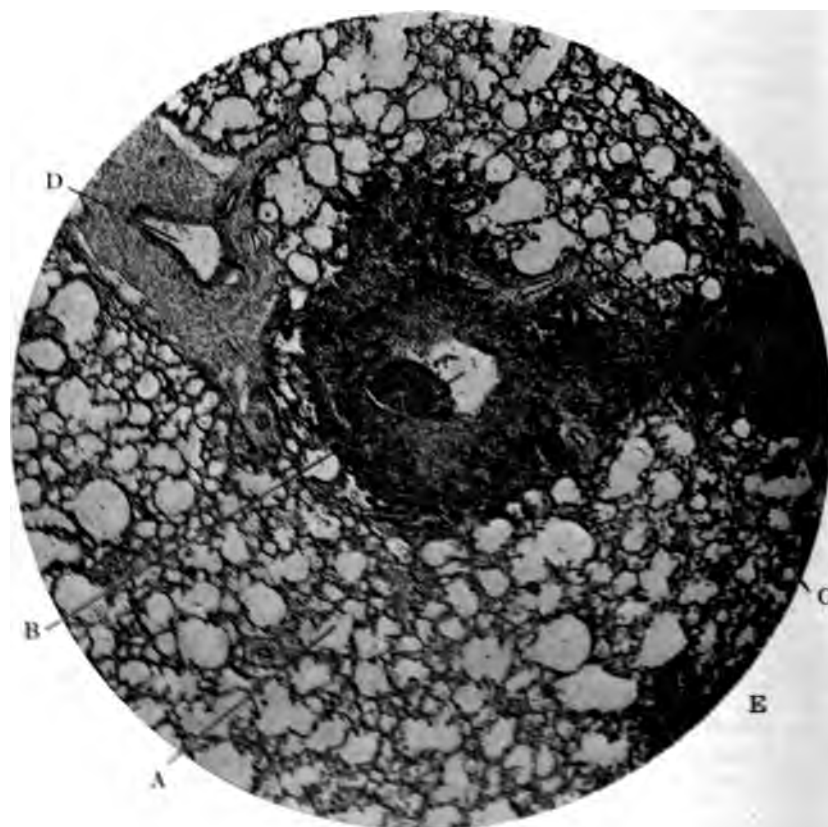
A large number of the patients had previously suffered from one or more attacks of bronchitis, and fifteen previously had broncho-pneumonia.

As an exciting cause, exposure to cold must still be classed among the potent factors of primary pneumonia. The organisms concerned in broncho-pneumonia have been discussed in the previous chapter.

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<sup>1</sup> It is probable that a number of cases complicating influenza were included among these primary cases.

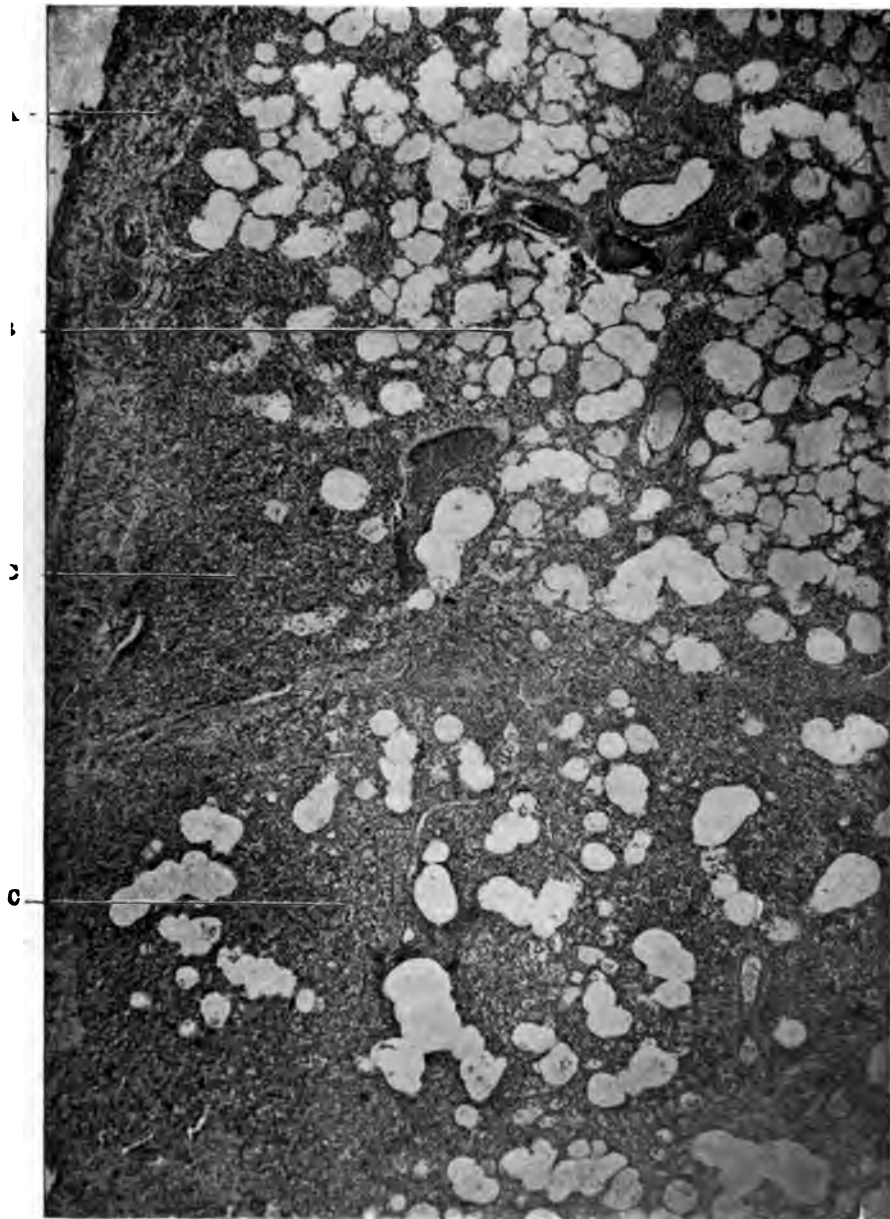
**Lesions.**—The term broncho-pneumonia is now generally adopted as a generic one, and it is to be preferred either to lobular or catarrhal pneumonia, as it gives prominence to the bronchial element in the inflammation. The process may begin in the larger tubes and gradually extend



**FIG. 72. BRONCHO-PNEUMONIA, WITH THICKENING OF A BRONCHUS.** In the centre of the picture is seen a small bronchus, B, which is cut somewhat obliquely; the degree to which its wall, C, is thickened is well shown. It is partially filled with pus, its mucous membrane is nearly destroyed, and its walls greatly thickened from infiltration with leucocytes. This infiltration extends to the lung tissue in the neighbourhood, it forms a per-bronchitic zone of pneumonia. Elsewhere in the picture the lung tissue, A, is practically normal. D is a small blood-vessel. E is another smaller bronchus. Throughout the lung everywhere surrounding the small bronchi similar changes were seen, in addition to which there were present some large areas of consolidation. The disease was of four and a half weeks' duration; the child, five months old.

It is seen in the accompanying micrograph that the secondary lobules in which pneumonia is situated are separated by normal lung vesicles which surround the bronchi. It is also seen that in whatever direction the small bronchi, zones of pneumonia (Fig. 72). In other cases the process seems to begin

PLATE XI.



ACUTE BRONCHO-PNEUMONIA.

Primary pneumonia in a child two years old, showing the irregular distribution of the consolidation and its incomplete character. A is the pleura somewhat thickened; B, lung tissue which is practically normal; C C are consolidated areas, scattered through which are groups of air vesicles still containing air. (Slightly magnified.)



almost at the same time in the small bronchi and the air vesicles, as both are found involved, even when death occurs within a few hours of the first symptoms.

There are, however, cases in which the parts of the lung affected bear no relation to the bronchi—where there are found simply smaller or larger areas of pneumonia irregularly scattered through the lung, usually near the surface (Plate XI). From the distribution of the lesions such cases might better be termed lobular than broncho-pneumonia.

Much has been said in the past about pulmonary collapse from obstruction of the small bronchi, as a condition antecedent to this form of pulmonary inflammation. So far as my own observations go, there has been adduced but little evidence that this is the rule, or, indeed, that it often occurs. Even in autopsies made very early in the disease, but little collapse is found, most of the cases supporting the view of Delafield, that when the disease extends from the bronchi to the air cells it involves those surrounding the tube quite as regularly as those to which the tube leads.

The following observations are made from a study of 170 autopsies of which I have records, microscopical examinations having been made in about one-third of the number.

*Seat of the Disease.*—In eighty-two per cent of the autopsies extensive disease was found in both lungs. The parts most affected were the lower lobes posteriorly; next to this the posterior part of both the upper and lower lobes. The left lower lobe was more extensively diseased than the right in over two-thirds of the cases. If the pneumonia is in front only, the right apex is the most frequent seat.

There are a certain number of cases which appear to follow tolerably well-defined stages of congestion, consolidation, and resolution; but the disease may be arrested at any of the stages and the case recover, or death may occur at any stage and there may be found at autopsy different portions of the lung representing all the stages mentioned. In considering, therefore, the lesions of broncho-pneumonia, it seems best to describe the condition in which the lungs are found at the various periods when death is likely to occur, rather than to attempt to describe the different stages of the disease, as in lobar pneumonia.

1. *The Acute Congestive Form (Acute Red Pneumonia).*—This is the condition in which the lung is usually found if death occurs during the first two or three days of the disease. In the cases severe enough to cause death in the first twenty-four hours, very little can be seen by the naked eye except acute congestion. The vessels of the pleura are distended, and there may be small superficial hemorrhages. Both lower lobes are usually heavy and dark coloured. There is to the naked eye no consolidation. All, or nearly all, the lung can be inflated. On sec-

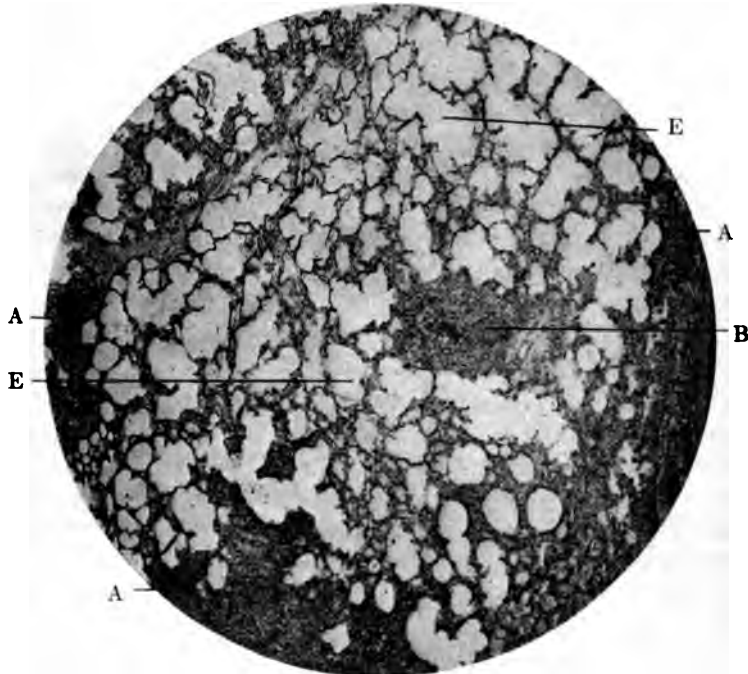
tion, there is found intense congestion with some œdema. When the process has lasted a little longer the affected areas are more sharply defined. These, usually the posterior portions of both lungs, are of a brownish-red colour, and appear partially consolidated, although with a little force they may in most cases be inflated. After section, pus and mucus flow from the divided bronchi, and the whole lung may be more or less congested or œdematous.

The microscope alone reveals the fact that these are not cases of simple pulmonary congestion or bronchitis of the finer tubes. In one case in which death occurred twelve hours from the first symptoms, I found well-marked evidences of inflammation of the air vesicles. In these hyper-acute cases, the microscope shows great distention of all the small blood-vessels of the affected area, and small or large extravasations of blood just beneath the pleura, into the alveoli and interstitial tissue of the lung. In some cases these hæmorrhages form the most striking feature of the lesion. The air vesicles are partially, some almost completely, filled with red blood-cells, swollen and desquamated epithelial cells, and a few leucocytes (Fig. 70). The red blood-cells predominate. The inflammation may be diffuse, involving nearly a whole lobe, or in small areas in the neighbourhood of the small bronchi. The mucous membrane of the large and small bronchi is the seat of catarrhal inflammation, and the walls of the latter are infiltrated with round cells.

When the process has lasted from twenty-four to forty-eight hours all the changes described are more marked, but the red colour of the inflammatory products still persists. Such cases give during life only the signs of congestion and bronchitis.

2. *The Mottled, Red and Gray Pneumonia.*—This is the usual appearance when the disease has lasted somewhat longer, and is found in most of the cases dying between the fourth and fourteenth days. There are usually at this time quite large areas of consolidation, sometimes affecting nearly an entire lobe, so that at first sight the case may resemble lobar pneumonia. This is sometimes described as the "pseudo-lobar" form. The extent of these areas depends largely upon the duration of the disease. In most cases there is pleurisy over the consolidated portions. This may cause the lung to adhere to the chest wall, the firmness of the adhesions depending upon the duration of the process. The surface of the lung is usually of a mottled red and gray colour; it often has a coarsely granular feel, due to the consolidation of some of the superficial lobules of the lung. On section, it is rarely found that an entire lobe is consolidated, the superficial portion being most affected, while the central part is normal or only congested. The colour is mottled, like that of the surface. In some places the consolidation appears complete; in others the consolidated areas are separated by healthy, congested, or emphysematous lung tissue (Fig. 73). The gray areas surround the

small bronchi and vary in size. The smallest ones look very much like miliary tubercles. The larger ones are seen where the process has existed for a longer time and has gradually invaded the contiguous air cells. If the lung is cut parallel with the bronchi, there may be seen small gray striæ of pneumonia along their course (Fig. 72, C). From the cut bronchi, pus flows quite freely on pressure. The bronchial walls are



**FIG. 73.—ACUTE BRONCHO-PNEUMONIA.** In the centre is shown a small bronchus, B, with a zone of pneumonia about it. The greater part of the section is made up of emphysematous lung tissue, E E, showing dilatation of the alveolar spaces and rupture of some of the alveolar septa. At the border, A A A, are seen the margins of consolidated areas of lung.

often seen to be thickened even by the naked eye. The parts affected are usually the posterior portions of the lower lobe of one or both sides, the remainder of the lobes being congested or œdematous, while in front the lung is emphysematous.

Under the microscope the smaller bronchi (Fig. 72) are seen to be much thickened and infiltrated with leucocytes. The gray areas surrounding the bronchi are made up of groups of air vesicles, which are packed with leucocytes (Fig. 74). Fibrin is sometimes seen in small amount, also red blood-cells and desquamated epithelial cells, but the leucocytes predominate. Surrounding the areas densely infiltrated are groups of air vesicles which are normal or congested, or which show only the earlier stages of the inflammatory process.



3. *Gray Pneumonia (Persistent Broncho-pneumonia)*.—This form is seen in protracted cases where there have been continuous symptoms usually for from three to six weeks. The pleuritic adhesions are more general and firmer. The amount of lung involved may be very great, often nearly the whole of both lungs posteriorly. The affected lung appears completely consolidated and slightly enlarged. On section, it is of a nearly uniform gray colour, sometimes of a yellowish-gray. On



FIG. 74.—BRONCHO-PNEUMONIA. Dense infiltration of pus cells in and about a small bronchus; under a low power. The cavity shown in the specimen is a cross-section of one of the small bronchi, which is partially filled with pus cells; the epithelium is destroyed. The bronchial wall and the pulmonary tissue in the neighbourhood are so densely infiltrated with leucocytes that almost every trace of normal structure is effaced. Child fifteen months old, disease of four weeks' duration. Extensive areas like this were found in both lungs.

pressure, pus exudes from the smaller and larger bronchi. The bronchial walls are markedly thickened, and in some places there may be a slight dilatation of the smaller bronchi. The part of the lung not consolidated may be almost white, owing to vesicular emphysema. In some cases there is also interstitial emphysema. Small cavities containing pus may

be found in the lung. The bronchial glands are frequently swollen to the size of a large bean, and are of a reddish-gray colour.

The microscope shows that the air vesicles of the consolidated portions are distended chiefly with leucocytes, but there are also epithelial

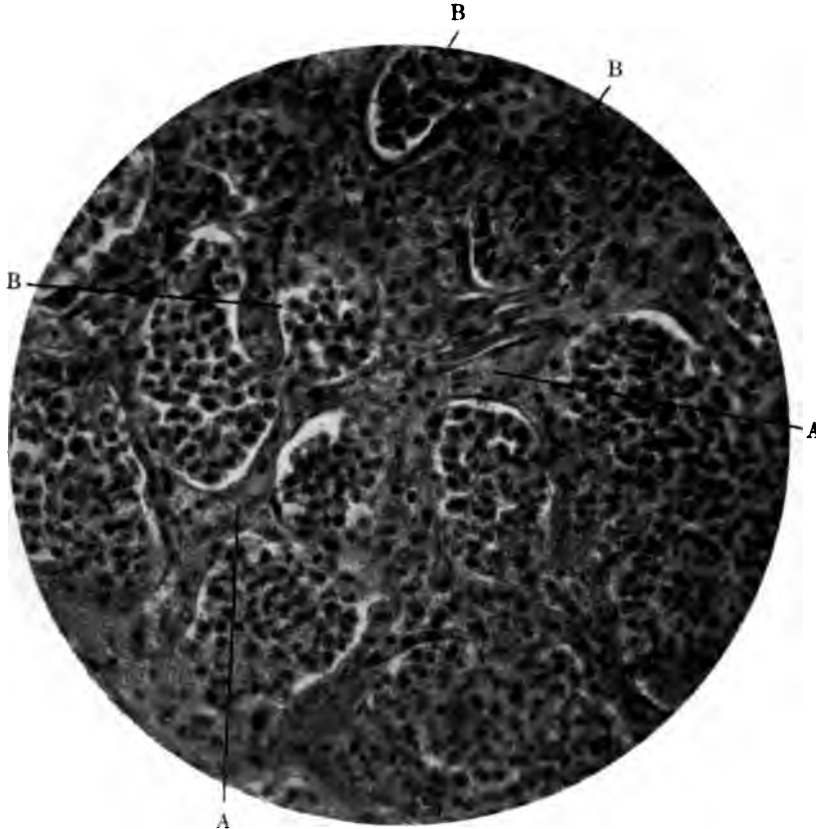


FIG. 75.—PERSISTENT BRONCHO-PNEUMONIA: HIGHLY MAGNIFIED. There is shown at A A marked thickening of the alveolar septa, encroaching upon the alveolar spaces. All the alveoli, B B, are densely packed with leucocytes. A similar condition also through nearly the whole of the affected lung. (For history and temperature, see Fig. 84.)

and connective-tissue cells. The alveolar septa may be so much thickened as to encroach upon the alveolar spaces (Fig. 75). Complete resolution is then impossible.

**Terminations.**—Death may occur at any stage, or the pathological process may be arrested at any stage and the case go on to recovery. Resolution may take place before any consolidation recognisable by physical signs has occurred; in such cases it is usually rapid and complete. If there has been consolidation, resolution may take place after two or three weeks and be complete, or it may be delayed for five or six weeks

and still be complete. In many cases, especially those in which it is delayed, resolution is only partial, and there are relapses or recurring attacks. After the first, or after several attacks, there may develop a chronic interstitial pneumonia; or simple pneumonia may be followed by tuberculosis. Such cases as these are to be carefully distinguished from the much more frequent ones in which the broncho-pneumonia is tuberculous from the outset.

**Associated Lesions of the Lungs.**—*Pleurisy* is almost invariably found over every large area of consolidation, and in cases of more than three or four days' duration; while in most of those fatal within the first two or three days the pleura is normal or only congested. It is seen in all grades of severity, from a slight gray film of fibrin that can hardly be stripped off, to a yellowish-green exudation one-fourth of an inch thick. A small amount of serum—one or two ounces—in the pleural sac is not uncommon, but a large serous effusion is very rare. Cases in which there is an excessive inflammation of the pleura are considered elsewhere under the head of *Pleuro-pneumonia*. *Empyema* occurs both during the stage of acute inflammation of the lung and while this is subsiding, but it is less frequent than in lobar pneumonia.

**Bronchial Glands.**—In all the recent acute cases these are swollen and red; the usual size is that of a pea or a bean. They show microscopically the usual changes of acute hyperplasia. In protracted cases, and after repeated attacks, they may be two or three times the size mentioned, and of a gray colour. It is rare that they are large enough to give rise to symptoms unless they become the seat of tuberculous deposits.

**Emphysema.**—This is one of the regular and striking features of acute broncho-pneumonia in infancy, it being especially marked in the protracted cases. It is usually vesicular, involving the greater part of the upper lobes in front and the anterior margin of the lower lobes. Occasionally interstitial emphysema is seen, forming either large striæ upon the surface of the lung, or blebs of considerable size along the anterior margin. This may occur even in cases uncomplicated by pertussis or laryngeal stenosis.

**Gangrene.**—Gangrenous areas were found in six cases of the series mentioned. In four of these the pneumonia was primary, in one it followed diphtheria, and in one ileo-colitis. It occurred in scattered areas of a grayish-green colour, varying from one-fourth of an inch to two inches in diameter.

**Abscesses of the lung** are by no means uncommon. They were noted in seven per cent of the autopsies. They are usually minute and multiple, varying in size from one-sixth to one-half inch in diameter. Sometimes a portion of a lobe is fairly honeycombed with minute abscesses. In one case a large abscess was found occupying the greater part of a lobe, the symptoms resembling those of empyema. Abscesses are usually

found in regions where the inflammatory process has been especially intense. They may be found in prolonged cases, in those of unusual severity, as shown by excessively high temperature and rapid extension of the disease, and in very delicate subjects. The microscope shows that these abscesses usually begin as an accumulation of pus in the small bronchi, whose walls become softened and break down on account of the intensity of the inflammation. They may be superficial, but are more commonly in the interior of the lung; they contain yellow pus and sometimes broken-down lung tissue. Small abscesses can not be recognised clinically; the large ones give the symptoms and signs of empyema. They are discussed more fully elsewhere. In several instances they have been successfully operated on, though wrongly diagnosticated.

The lesions in other organs will be considered under Complications.

**Symptoms.**—Broncho-pneumonia has no typical course. The cases differ from each other very markedly, but they may be divided into a few quite distinct groups.

1. **THE ACUTE CONGESTIVE TYPE.**—This may be seen at any age, but is more frequent in young infants. It may be either primary or secondary, being not uncommon in either form. Its symptoms are few and irregular, and the disease is often unrecognised. The entire duration may be only twenty-four hours. High temperature, extreme prostration, cyanosis, and rapid respiration may be the only symptoms. The temperature varies between 104° and 107° F., usually rising steadily until death occurs. The prostration is extreme from the outset, the patient being overwhelmed by the suddenness and severity of the attack. Cyanosis is frequently present, and is almost always seen shortly before death. The respirations are from 60 to 80 a minute, but in most cases not strikingly laboured. Cough is frequently absent. Cerebral symptoms are often marked. There are dulness and apathy, sometimes quite profound stupor, and not infrequently convulsions just before death. The physical signs are few and inconclusive. There is often nothing abnormal except very rude breathing over both lungs behind; sometimes the breathing on one side is feeble, and on the other much exaggerated. There may be no râles whatever, and no change in the percussion note.

The suddenness and severity of these symptoms are something which it is hard for one who has not observed them to appreciate. I have known an infant to die in twelve hours from the time in which he was apparently in perfect health, and had an opportunity to confirm the diagnosis of pneumonia by a microscopical examination of the lung. The diagnosis can not be positively made during life, and in most of the cases the disease passes under some other name. It is often regarded as malignant scarlet fever or measles with suppressed eruption, or cerebro-spinal meningitis.

If the children are sufficiently strong to withstand the onset of vio-

lent symptoms, they may recover completely in four or five days, the lung clearing up very rapidly. In other cases these grave symptoms may abate in a day or two, to be followed by those of ordinary broncho-pneumonia, which runs its usual course.

The symptoms of some of these cases may be explained by the sudden intense engorgement of the lung, which, owing to the small size of the air vesicles, interferes with its function almost as much as does consolidation. In other cases the symptoms are due not so much to the pulmonary condition as to a general pneumococcus infection. A case lately came under my notice in which death occurred after a thirty hours' illness, where the pneumococcus was found by culture in both kidneys, the spleen, heart's blood, and both lungs.

2. ACUTE DISSEMINATED BRONCHO-PNEUMONIA (CAPILLARY BRONCHITIS).—Although the symptoms in this class of cases are chiefly due to the bronchitis, I have never failed to find at autopsy evidences of pneumonia also. These are not very common cases. The process begins as an inflammation of the medium-sized and small bronchi, but not of the finest bronchi. The onset is acute, with fever, very rapid and laboured breathing, severe cough, moderate prostration, and in most cases cyanosis.

The temperature is not high, usually only from 100° to 102° F., and it often continues so for three or four days. The pulse is rapid, and at first is full and strong. The respirations are exceedingly rapid, often from 80 to 100 a minute. There is dyspnoea with marked recession of all the soft parts of the chest during inspiration. Cough is always present, usually severe, and sometimes almost incessant. The prostration is not so great as in the cases previously described, and the development of the symptoms is much less rapid.

There are at first sibilant and afterward subcrepitant râles over the entire chest, with which are usually mingled coarser moist râles. There are no evidences of consolidation. The respiratory murmur is everywhere feeble, but not otherwise altered. Percussion generally gives exaggerated resonance, owing to the emphysema which is present, the note being sometimes almost tympanitic.

The symptoms may gradually increase in severity until death takes place by the third or fourth day, from respiratory or cardiac failure. There is usually marked cyanosis, and toward the end rapidly increasing prostration. Just before death the temperature often rises rapidly to 106° or 107° F. At the autopsy there are found evidences of bronchitis of the tubes of all sizes, and minute zones of pneumonia about the smaller bronchi. The lungs are generally in a state of hyper-inflation, on account of which they do not collapse on opening the chest. There may be in addition extensive congestion or œdema, the development of which has been the immediate cause of death.

In cases which do not prove fatal there is usually by the third or fourth day great improvement in the general symptoms; the finer râles may disappear, and the coarse ones become more and more prominent. By the end of a week there may be complete recovery. Instead of this, there may be a continuance of the constitutional symptoms, and disappearance of the fine râles in front only, while behind there are gradually added to them the signs of consolidation in one of the lower lobes near the spine. From this time the case may progress as one of ordinary broncho-pneumonia.

The prognosis in this class of cases is very much better than in the congestive variety, recovery being probable unless the patients are very young or very delicate infants.

3. BRONCHO-PNEUMONIA OF THE COMMON TYPE.—When primary, this usually begins suddenly with symptoms not unlike those of lobar pneumonia. This was the mode of onset in two-thirds of my cases. In only ten per cent was the pneumonia preceded by bronchitis of the large tubes. In these the symptoms of bronchitis may slowly or rapidly (Fig. 76) merge into those of pneumonia. When the onset is sudden it is marked by high fever, frequently by vomiting, rarely by convulsions. In addition there are rapid respiration, cough, prostration, and sometimes cyanosis. The symptoms are more distinctly pulmonary than is generally the case in lobar pneumonia.

The temperature, as a rule, is high; rarely is it continuously so, but it is of a remittent type. The daily fluctuations often amount to four or five degrees. The fever usually continues from one to three weeks, and gradually subsides. It is rare for it to terminate by crisis. Although, as a rule, we expect a high temperature with acute pneumonia, this is not invariable. Primary cases may run their course, and even terminate fatally, although the temperature has not been above 101° F. I have records of several such cases. A low temperature is more often seen in young and delicate infants than in those who are older and more robust.

The respirations are frequent and laboured; there is real dyspnoea. On inspiration, there are marked recessions of all the soft parts of the chest, and the alæ nasi dilate actively. The usual rapidity of the respirations is from 60 to 80 per minute; very often, however, it rises to 100, and on several occasions I have seen it even 120. Respiration generally seems more embarrassed than does the action of the heart, and respiratory failure is a more frequent cause of death than cardiac failure. The pulse is always rapid—from 150 to 200 a minute—and when so it is often irregular. The pulse rate is of much less importance than its character. Early it is full and strong, but soon it becomes soft, compressible, and weak.

The prostration is usually moderate for the first day or two, but

steadily increases as the lung becomes more and more involved, and toward the close of the disease may be extreme.

Cough is much more constant than in lobar pneumonia, and more distressing; sometimes it is almost incessant. It disturbs rest and sleep, and may cause vomiting if the paroxysm occurs soon after eating. There is no expectoration. Mucus is sometimes coughed up into the trachea, or even the pharynx, to be swallowed again, or more frequently aspirated into the lung. If during a severe paroxysm the patient is turned upon his face or inverted, much of this mucus may be dislodged. A strong cough is a good symptom; suppression of the cough is a bad symptom, indicating a loss of the reflex sensibility of the bronchial mucous membrane and of the respiratory centre.

Pain in the chest is not common, and is rarely an annoying symptom.

Cyanosis is present at some time in most of the severe cases. It may occur at the onset, or at any time during the course of the disease. It is usually due to sudden congestion of a portion of the lung not previously involved. Even when slight, it is always a danger-signal of respiratory failure, and when present only in the finger tips or lips indicates that the patient must be carefully watched and energetically treated. In the severe cases the whole body may be of a dull leaden hue.

Nervous symptoms at the onset are not so frequent as in lobar pneumonia, convulsions being rare; but late convulsions, particularly in the pneumonia which complicates pertussis, are frequent, and when present the disease is usually fatal. Delirium may occur at any time during the attack. In infants this shows itself by excitement and inability to recognise the nurse or mother. Occasionally patients present marked cerebral symptoms throughout the disease closely simulating meningitis. As elsewhere stated, the nervous symptoms depend not upon the location of the disease, but upon its extent, the intensity of the infection, and upon the susceptibility of the patient, such symptoms being especially common in rachitic children and in those suffering from pertussis.

Gastro-enteric symptoms are frequent in infancy, and are of much importance. Often there are from four to six stools a day, of a green colour, containing mucus and undigested food. These symptoms depend upon the feeble digestion which is associated with the febrile process, and are often aggravated by improper feeding and overmedication. Vomiting and diarrhoea add much to the danger of the attack. In summer this complication is more frequent and is likely to be more severe. Distention of the stomach or intestines from gas may be the cause of distressing symptoms, owing to the added embarrassment of respiration produced by this upward pressure. In infants it may lead to attacks of cyanosis, and even to convulsions.

The blood in acute broncho-pneumonia shows regularly the changes

of a moderate secondary anæmia, which in protracted cases becomes very marked. A leucocytosis is almost invariably present. In an average case this ranges from 20,000 to 40,000. It sometimes is excessively high without any apparent reason. I have several times seen it over 100,000. The increase is chiefly in the polymorphonuclear cells which usually form from sixty to eighty per cent of the total leucocytes. With the fall in temperature the leucocytosis in most cases rapidly disappears. A rapid diminution in the leucocytosis may indicate a marked loss of resistance in the patient; and may be seen with either a high or a low temperature. In the pneumonia which complicates pertussis, the increase in the white cells is chiefly of the lymphocytes.

The urine in most cases is scanty, high-coloured, and loaded with urates. A trace of albumin is often present when the temperature is very high; but casts, renal epithelium, and a large amount of albumin are rare.

The accompanying temperature chart (Fig. 76) is a good example of a very frequent course of primary pneumonia of moderate severity terminating in recovery. In cases of this type the constitutional symptoms are not grave, and follow very closely the temperature curve.

The next chart (Fig. 77) illustrates a more severe but not uncommon course of the disease in which the fever is prolonged. The usual

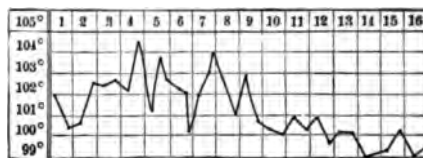


FIG. 76.—TEMPERATURE CURVE IN TYPICAL BRONCHO-PNEUMONIA OF THE Milder FORM. *History.*—Male, sixteen months old; delicate child; previous bronchitis; onset gradual; signs of consolidation at left base on fifth day, but fine râles over both lower lobes behind; resolution slow, râles persisting for a long time in both lungs.

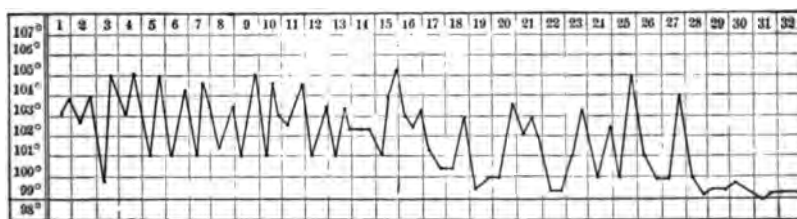
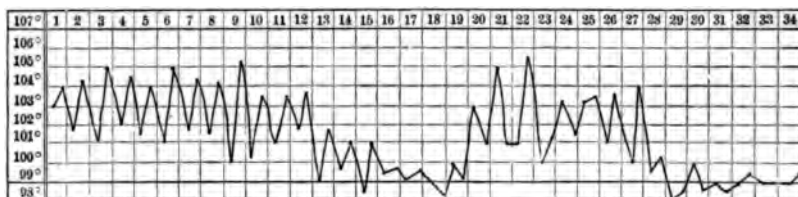


FIG. 77.—TEMPERATURE CURVE OF BRONCHO-PNEUMONIA WITH A PROLONGED COURSE; RECOVERY. *History.*—Female, eighteen months old; in fair condition; sudden onset. Early signs were localised, fine râles over left base; on fifth day signs of consolidation at left base, with râles on both sides behind. General symptoms of moderate severity. Signs of consolidation disappeared about a week after cessation of fever; râles persisted nearly two weeks longer.

duration of cases of this type is between three and four weeks. The irregular fluctuations of the temperature, rarely touching the normal line, are exceedingly characteristic of broncho-pneumonia.

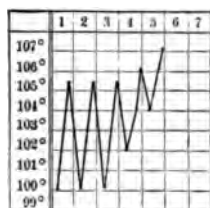


The chart shown in Fig. 78 is that of relapsing pneumonia. The first attack was fairly typical, with about the usual duration. Resolu-



**FIG. 78.—TEMPERATURE CURVE OF RELAPSING BRONCHO-PNEUMONIA; RECOVERY.**  
*History.*—Male, nineteen months old; delicate. Consolidation on sixth day in left lower lobe behind; two days later small area of consolidation in right lower lobe behind; many râles both sides; eighteenth day, signs of consolidation had disappeared, but many râles persisted. Accession of fever on nineteenth and twentieth days, accompanied by extension of disease as shown by new râles, but no evidences of consolidation during second attack. Slow resolution and convalescence.

tion had begun, and was apparently progressing favourably, when there was a return of the fever, accompanied by new signs in the chest, the



**FIG. 79.—TEMPERATURE CURVE OF BRONCHO - PNEUMONIA; FATAL.**  
*History.*—Male, six months old; markedly rachitic; sudden onset. Signs first day were fine moist râles throughout the chest, marked prostration, and cyanosis; on third day, a small area of consolidation in upper lobe of left lung behind; increasing prostration, cyanosis, and death.  
*Autopsy.*—No pleurisy; consolidation at left apex behind, and posterior two-thirds of left lower lobe; consolidation of right apex posteriorly, lower lobe intensely congested.

second attack being shorter and milder than the first. Very often the temperature falls to normal without any signs of resolution, and after an interval varying from two or three days to a week there is a recurrence of the fever and other constitutional symptoms, the second attack frequently proving fatal.

A frequent course in fatal cases is shown in Fig. 79. The duration of the disease, instead of being five days as in this case, is often only three or four. The temperature at first fluctuates widely, then rises gradually until death.

*Duration of the Fever.*—The following figures give the duration of the fever in 231 cases. The majority were primary; none were secondary to diphtheria, and only a few complicated measles. Of the 169 cases that were fatal—

|  |                 |
|--|-----------------|
| There died during the first six days . . . . .         | 25.0 per cent.  |
| “ “ between the seventh and twenty-first days. . . . . | 55.5 “ “        |
| “ “ “ “ twenty-first and sixtieth days. . . . .        | 19.5 “ “        |
|  | <hr/> 100.0 “ “ |

Of 78 cases which recovered, the duration of the fever was—

|                                     |                |
|-------------------------------------|----------------|
| Less than seven days .....          | 11.5 per cent. |
| From seven to twenty-one days ..... | 66.6 " "       |
| " twenty-one to ninety days .....   | 21.9 " "       |
|                                     | <hr/>          |
|                                     | 100.0 " "      |

*Physical Signs.*—In considering the signs of broncho-pneumonia, it is better to connect them with the different conditions in the lung than to group them in stages, as in lobar pneumonia.

(a) *Without Consolidation.*—It can not too often be repeated that broncho-pneumonia may exist without signs of consolidation at any period during the course of the disease. When the attack is primary, the earliest signs are due to congestion of the lung, associated with bronchitis of the fine tubes, which is usually localised, but which may be general. If the disease has followed bronchitis of the large tubes, its signs are added. Congestion of the lung gives feeble breathing over the affected area, and occasionally slight dullness or diminished resonance. With this are found coarse sonorous, and finer sibilant râles, due to congestion and swelling of the mucous membrane of the larger and smaller bronchi respectively. These signs are soon replaced by very fine moist râles, which are usually localised in one of the lower lobes behind (Fig. 80). These localised fine râles are the first distinctive sign of broncho-pneumonia. Soon a change in the respiratory murmur is heard in the affected area, which becomes feebler in intensity and higher in pitch. Elsewhere in the chest there may be coarse râles, due to bronchitis of the large tubes. In such cases the areas of pneumonia are so small and so scattered as to give in themselves no additional signs, and the case may go on to recovery without presenting anything more distinctive than the signs mentioned.

(b) *With Areas of Partial Consolidation.*—In the lung at this time there are small areas of consolidation, generally superficial and separated by healthy or congested lobules. Percussion in these cases usually gives negative results, but sometimes there is very slight dullness. The vocal fremitus is not usually altered. The fine moist râles may be heard over quite a large area, but at some point, usually near the spine, over one of the lower lobes, they are sharper, louder, higher pitched, and more metallic, and seem close under the ear (Fig. 81). Respiration is feebler here than elsewhere, and broncho-vesicular in quality, approaching bronchial breathing more and more as the consolidation increases. The resonance of the voice and cry is exaggerated.

(c) *With Areas of Consolidation More or Less Complete.*—On percussion there is dullness, but surprisingly little in comparison with the other signs of consolidation present. It is due to the fact that the consolidated portion, though extensive, does not involve the lung to any great depth, and also that there are in the consolidated area many alveoli which still contain air (Plate XI). On palpation there is usually a slight increase in the vocal fremitus. On auscultation, there



FIG. 80.—FIRST STAGE. Coarse râles over both lungs; localised fine (subcrepitant) râles at the left base. No change in breath sounds.



FIG. 81.—SECOND STAGE. Coarse and fine râles over both lungs behind; at left base an area of partial consolidation, with broncho-vesicular breathing, exaggerated voice, and very sharp râles.



FIG. 82.—THIRD STAGE. A larger area of partial consolidation, and in the centre a small area of complete consolidation, with bronchial breathing and voice and slight dulness. Signs over the right lung similar to what were previously present over the left.



FIG. 83.—FOURTH STAGE. Extensive disease of both sides; large area of complete consolidation on the left, with dulness, bronchial breathing and voice, and no râles; surrounding this, broncho-vesicular breathing, with many râles. Signs in the right lung similar to those previously present over the left.

NOTE.—The large circles indicate coarse râles; the small ones finer râles; the red areas indicate consolidation partial or complete. The disease may stop at any one of these stages and resolution take place.

are still present the evidences of bronchitis, usually only behind, but sometimes over the entire chest. Coarse and fine râles are intermingled. Over the consolidated parts are heard bronchial breathing and bronchial voice. At the centre of these areas the bronchial breathing is pure and râles are usually absent, but at the margin râles are present and the breathing approaches the broncho-vesicular type (Fig. 82). The signs of consolidation are rarely sharply circumscribed as they are in lobar pneumonia, but shade off gradually. The consolidated area is at first small, usually in one of the lower lobes near the spine, but may gradually extend until nearly the whole of one or even both lungs behind are more or less completely solidified (Fig. 83). The signs are found as far forward as the axillary line, but usually stop there. Friction sounds may be heard over the consolidated areas, but very rarely except where signs of complete consolidation are present. It is often impossible to obtain any idea of the condition of an infant's lung during quiet, superficial respiration. Sometimes over a part which is completely consolidated there is heard only very feeble breathing, or the lung may be almost silent. If, however, the child is made to cry or to take a deep inspiration, both the bronchial breathing and râles are distinctly brought out. The intensity of the consolidation increases as the case advances, and the signs become more and more like those of lobar pneumonia. During resolution there is first a disappearance of the signs of consolidation, which may be quite rapid, but friction sounds and râles of all kinds often persist for three or four weeks longer.

The following statistics are of some interest, as showing the frequency with which signs of consolidation were found, and the day when they were discovered. Their value is increased by the fact that the children were under observation in an institution at the time they were taken sick, and that in all the fatal cases—thirty-six in number—in which signs of consolidation were absent, the diagnosis of pneumonia was confirmed by autopsy:

|  |             |
|--|-------------|
| Consolidation noted on or before the fourth day..... | 47 cases.   |
| “ “ from the fifth to the seventh day.....           | 36 “        |
| “ “ “ the eighth to the twelfth day....              | 12 “        |
| “ “ after the twelfth day.....                       | 9 “         |
| No signs of consolidation.....                       | 62 “        |
|  | <hr/> 106 “ |

In general, it must be borne in mind that in many cases signs of consolidation are never present, as the areas of pneumonia are small and widely scattered; that where there is consolidation it is usually incomplete, because there are small areas of healthy lung tissue between the hepatised portions; that the signs of consolidation usually shade off gradually; and that both sides are almost invariably involved, although one side usually to a greater degree than the other.

4. THE PROTRACTED FORM—PERSISTENT BRONCHO-PNEUMONIA.—This is seen in primary cases, especially among delicate children, and in the pneumonia complicating pertussis, influenza and measles, and is the form which often follows diphtheria. The onset and course of the disease for the first two or three weeks do not differ from an ordinary attack of moderate severity, but at the end of this period there is seen no tendency in the process to subside. The fever continues, although it may not be high, but by physical examination it is found that the areas of consolidation are gradually increasing day by day, until sometimes the greater part of both lungs behind are involved. The air vesicles become so distended with cells that the signs of consolidation are more complete than in ordinary broncho-pneumonia. There is marked dulness, sometimes almost flatness; bronchial breathing is exaggerated in intensity, until it resembles cavernous breathing, and it may be impossible to distinguish between them. However, the fact that it is heard over so large an area, that it shades off gradually, and that it is accompanied by friction sounds, usually make a distinction possible.

The temperature in these protracted cases for the first two or three weeks is from 100° to 105° F.; but after this time it is generally lower—from 100° to 102° or 103° F. The course is not at all regular, but

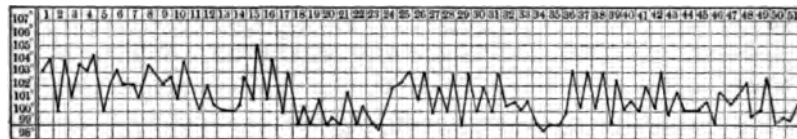


FIG. 84.—TEMPERATURE CURVE OF PERSISTENT BRONCHO-PNEUMONIA, TERMINATING FATALLY. *History.*—Male, two and a half years old; healthy; sudden onset; for two weeks the only signs were very fine moist râles throughout both lungs, front and back. The râles in front in great part gradually cleared up; those behind persisted, but it was not until the thirty-fourth day that positive signs of consolidation were discovered in the left lower lobe behind; these signs gradually extended, and, before death, were present over nearly the whole left lung behind and over the right lower lobe. There were also friction sounds over both lungs. *Autopsy.*—Old and recent pleurisy with general adhesions; left lower lobe completely solid, patches of consolidation in left upper lobe. Right lower lobe about one-half consolidated, with patches elsewhere. Bronchial glands large, but not cheesy. No evidence of tuberculosis upon either gross or microscopical examination (see Fig. 75).

marked by frequent exacerbations and remissions. The general symptoms are those of progressive asthenia. There is continued wasting, anæmia, and steadily increasing prostration. The appetite is lost, often there is an aversion to food, and vomiting is easily excited if food or stimulants are forced. The stools show that even what food is taken is very imperfectly digested and assimilated. The skin becomes dry and loses its elasticity; bed-sores may form; fine punctate hæmorrhages are seen over the abdomen, sometimes over the chest and extremities. The latter is

always a very bad symptom, and I have never seen recovery from pneumonia when it was present.

Death takes place from slow asthenia, usually after five or six weeks, but the attack may be prolonged for eight or ten weeks. The general symptoms, the temperature, and the wasting strikingly resemble cases of tuberculosis, and such is the diagnosis often made.

Although the majority of the cases in which the fever lasts over four weeks run the fatal course just described, such apparently hopeless cases occasionally recover. The temperature gradually falls lower and lower, until it remains at the normal point. For some time after this, often two or three weeks, little change can be seen, either in the general symptoms or in the physical signs. Gradually the appetite returns, the child is brighter and begins to take an interest in its surroundings, the cough abates, and little by little the signs in the lungs clear up, and the case may go on to complete recovery. Convalescence, however, is always slow, and may be interrupted by relapses, it being many months before health is fully restored. Although the signs of consolidation disappear in a few weeks, râles are apt to persist for a much longer time. It is probable in such cases, even though all signs of disease disappear from the chest, that the lung does not become quite normal, and relapses and second attacks are always possible. The general health may be so undermined that the child never regains his former vigour; yet in a surprising number of these cases recovery seems to be complete. Protracted cases of a mild type are sometimes seen, and, although the temperature persists for a number of weeks, it is never high. The course of the disease suggests tuberculosis. One such case in a young infant under my care was due to a staphylococcus infection, and was cured by vaccines.

##### 5. SECONDARY PNEUMONIA.

###### —(a) *Complicating Pertussis.*—

It is not often that pneumonia develops during the first two weeks of this disease. The most frequent time is from the third to the fifth week, when the patient has become exhausted from the previous severity of the pertussis. In two-thirds of my cases the development of the pneumonia was gradual, following bronchitis of the larger tubes. The temperature chart shown in Fig. 85 well illustrates this course.

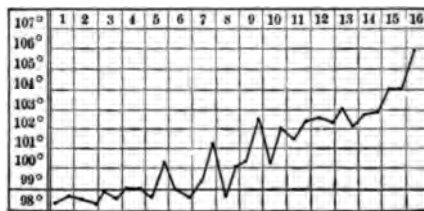


FIG. 85.—TEMPERATURE CURVE OF FATAL BRONCHO-PNEUMONIA, COMPLICATING PERTUSSIS. *History.*—Male, six months old; delicate; pertussis for three weeks. Early signs of bronchitis of large tubes only; on the eleventh day signs of consolidation in right upper lobe. Increasing prostration, cyanosis, and death. *Autopsy.*—Large areas of consolidation in right middle and upper lobes, small scattered spots throughout left lung.

When the onset is sudden, the symptoms do not differ essentially from those of primary pneumonia. The temperature of pertussis-pneumonia is usually not high, in a very large number of cases not rising above 103.5° F., and ranging most of the time from 101° to 103° F. These cases are very apt to be prolonged, the fever often lasting for three or four, and sometimes even for six, weeks. The physical signs of consolidation may persist for a long time after the temperature has become normal, and yet the case may recover entirely. I have seen one case in which complete recovery occurred after the signs of consolidation had persisted for six months, and another in which they had persisted for over eight months. Very often the signs continue during the entire attack of pertussis. Cerebral symptoms are common, especially toward the close of the disease. Of fifty-four fatal cases, twenty-five had convulsions, and in twenty-two this was the mode of death. Only one case which developed convulsions recovered.

(b) *Complicating Measles.*—In a small number of cases the pneumonia begins simultaneously with the invasion of measles, but generally not until the eruption appears. Instead of gradually falling to normal with the fading of the eruption, the temperature continues high. Any of the clinical types of primary pneumonia may occur in measles, the acute congestive variety, which is fatal in two or three days, being especially common. In its course and duration the pneumonia of measles resembles the severe form of primary pneumonia. The broncho-pneumonia of scarlet fever differs in no way from that of measles.

(c) *Complicating Diphtheria.*—In many cases this does not give a distinct clinical picture of its own, its symptoms being mingled with those of diphtheritic bronchitis, with which it is frequently associated. In others the forms resemble those seen in measles. The majority of cases occur as a complication of diphtheria of the larynx, although it is not infrequent in the septic cases in which only the upper air passages are involved. Pneumonia after laryngitis may develop within two days from the beginning of laryngeal symptoms, and run a rapid course; or it may come as late as the second or third week. In a child wearing a tube, the diagnosis of pneumonia presents difficulties, owing to the alteration in the respiratory sounds and the existence of the loud tracheal râles which obscure the usual auscultatory signs. Although pneumonia may be apparent by symptoms, its situation may be difficult to determine. The most important signs for diagnosis are the diminished respiratory murmur, localised râles, and dulness on percussion.

(d) *Complicating Influenza.*—Without doubt many cases usually regarded as primary are really secondary to influenza, particularly when that disease is prevalent. While the pneumonia of influenza may differ

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in no essential points from the primary form, there are types which are quite characteristic. In one variety the cases are of short duration, frequently lasting but three or four days, but with high and often widely fluctuating temperature, the general symptoms being of only moderate severity. A second type is a prolonged pneumonia with exacerbations and remissions, which may last for two or three months. A third form is the recurrent type of pneumonia, of which a child will sometimes have three or four attacks in a single season, separated by several weeks in which a moderate cough and a few coarse râles in the chest are the only signs of disease.

(e) *Complicating Ileo-colitis.*—This is usually a somewhat subacute form of pneumonia which is scarcely recognisable except by the physical signs. It is seen in the protracted cases of ileo-colitis, usually the ulcerative variety, and occurs late in its course. Very often pneumonia is not suspected during life, the constitutional symptoms being sufficiently explained by the intestinal lesions, although the autopsy discloses the fact that death was due in part to pneumonia.

**Complications.**—Most of those relating to the lungs have been described with the lesions. Pleurisy will be separately considered. Pulmonary emphysema is always present to a greater or less degree, but can not be made out by physical signs. In very rare instances subcutaneous emphysema has been seen. Abscess and gangrene can seldom be recognised by physical signs. Pneumothorax occurs even in infancy, but is very infrequent. Otitis is exceedingly common, and one should be constantly on the lookout for it. It is recognised only by examination of the ear with a speculum.

Meningitis may complicate acute broncho-pneumonia. It has occurred in about two per cent of my cases. It is in all respects similar to that occurring with lobar pneumonia. Meningeal hæmorrhage I have seen only once, and was the cause of death in a patient eleven months old, who a few days before was seized with convulsions, followed by a gradually increasing stupor, which continued until death. The hæmorrhage covered the entire convexity of the brain. Endocarditis is extremely rare; it was not observed in any of my cases. Acute pericarditis is also rare, and when it occurs it is usually with pneumonia of the left side. Complications referable to the digestive tract are quite common. Herpetic stomatitis is frequent, and occasionally the ulcerative variety is seen. Thrush often occurs in the protracted cases among very young infants. Gastro-enteritis is not very common, considering the frequency of vomiting and diarrhœa, these depending usually upon functional derangement. In only three of my cases was there nephritis. In all it was of the acute exudative variety, and in only one case was it severe enough to affect the prognosis.

Old lesions of tuberculosis—cheesy nodules in the lungs and some-



times in the pleura—are not infrequently met with in patients dying of acute pneumonia of a non-tuberculous character.

**Diagnosis.**—An acute onset with continuous high fever, rapid respiration, and cough, should always lead one to suspect pneumonia. When to these symptoms are added prostration and a leucocytosis, the diagnosis of pneumonia is almost certain. Cases of the acute congestive type are the ones most frequently unrecognised, and in many of these cases a positive diagnosis is impossible during life. Many atypical cases of pneumonia are seen, particularly in young infants. An unusual temperature course is perhaps the symptom most likely to lead to a mistake. While this, as a rule, is high and remittent, it is sometimes not so, and may be but little above normal. Rapid respiration is almost always present, but cough may be very slight, especially in infants. In very young infants, the diagnosis often rests upon the prostration, cyanosis, and rapid respiration, the other acute inflammatory symptoms being absent. Only the physical signs of the disease can positively settle the question of diagnosis.

When pneumonia follows bronchitis of the large tubes, whether the bronchitis is primary or complicates one of the infectious diseases, the extension of the disease to the lungs is usually marked by three symptoms—a steadily rising temperature, more frequent respiration, and increasing prostration. It may be twelve or twenty-four hours before the change is indicated by the physical signs.

The diagnosis of broncho-pneumonia from congenital atelectasis has to be considered only during the first three or four months of life, it being rare for atelectasis to give symptoms after this time. In early infancy the danger of confusing the two is increased by the fact that atelectasis and broncho-pneumonia may be associated. If the infant has been strong and well for the first two months, congenital atelectasis can be excluded. It is likely to be found in delicate infants, where there is a history of difficulty in resuscitation at birth and feeble cry during the early days of life. The temperature is low, often subnormal, the cyanosis is out of proportion to the other symptoms, and the physical signs are doubtful or absent.

At the outset, pneumonia can not be positively diagnosticated from severe bronchitis. Such a bronchitis often begins with severe pulmonary symptoms and a temperature of 103° or 104° F.; but this high temperature is of short duration, usually falling after twenty-four or forty-eight hours to 100° or 101° F. The prostration is much less and all the symptoms, possibly excepting the cough, less severe. The only physical signs are coarse râles, which are heard throughout the chest.

The same rules apply to bronchitis of the smaller tubes. The râles are heard both in front and behind, and usually over both sides. If with such râles the temperature continues to rise for three days in succession

above 103° F., it may be assumed that pneumonia is present, provided there is no other disease which might explain the temperature. If, instead of being generalised, the signs of bronchitis are limited to a single lung, or to one lung posteriorly, the existence of broncho-pneumonia may be regarded as certain. Localised bronchitis, then, is always to be interpreted as broncho-pneumonia, provided tuberculosis can be excluded. In doubtful cases the chances largely favour broncho-pneumonia rather than bronchitis. Attention is again called to the fact already mentioned, that there are a large number of cases of pneumonia without signs of consolidation.

The differential diagnosis of broncho-pneumonia from lobar pneumonia will be considered in connection with the latter disease. On account of the remittent temperature, broncho-pneumonia may be confounded with malarial fever; or malaria may be suspected as a complication. An examination of the blood will remove the doubt.

Both the acute and the persistent forms of simple broncho-pneumonia may be confounded with the tuberculous form; the points of distinction are considered in the chapter on Tuberculosis.

**Prognosis.**—Broncho-pneumonia is always a serious disease, and in an infant dangerous to life. The prognosis depends upon the age, surroundings, and previous condition of the patient, upon the nature of the infection, whether the disease is primary or secondary, and, if the latter, upon the character of the primary disease. In private practice the mortality from broncho-pneumonia is from ten to twenty per cent, depending upon the conditions mentioned. One whose knowledge of broncho-pneumonia is derived from observations in private practice can, however, form but little idea of the frequency and severity of this disease in hospitals and asylums for infants and young children, particularly when it occurs with epidemics of measles, diphtheria, and pertussis. The statistics in the following table are taken from the records of two institutions with which I was at the time connected, and fairly represent the results seen in such places in children under three years:

| FORMS OF PNEUMONIA.                         | Cases. | Deaths. | Percentage mortality. |
|---|--------|---------|-----------------------|
| Primary broncho-pneumonia . . . . .         | 194    | 96      | 49.4                  |
| Following bronchitis of the large tubes . . | 29     | 19      | 65.5                  |
| Secondary to measles . . . . .              | 89     | 56      | 62.9                  |
| “ “ pertussis . . . . .                     | 66     | 54      | 81.8                  |
| “ “ scarlet fever . . . . .                 | 7      | 7       | 100.0                 |
| “ “ diphtheria . . . . .                    | 47     | 47      | 100.0                 |
| “ “ ileo-colitis . . . . .                  | 19     | 18      | 94.7                  |
| “ “ epidemic influenza . . . . .            | 8      | 1       | 16.6                  |
| “ “ varicella . . . . .                     | 2      | 2       | 100.0                 |
| “ “ erysipelas . . . . .                    | 2      | 2       | 100.0                 |
| Totals . . . . .                            | 461    | 302     | 65.5                  |

The mortality varies with the age of the patient, being highest during the first year, and diminishing steadily thereafter, as shown by the following table giving the result in 346 cases:

| Age.                        | Cases. | Percentage mortality. |
|-----------------------------|--------|-----------------------|
| During the first year ..... | 202    | 66                    |
| " " second year .....       | 102    | 55                    |
| " " third " .....           | 33     | 33                    |
| " " fourth " .....          | 6      | 16                    |
| " " fifth " .....           | 3      | ..                    |

In this table are included no cases secondary to measles, scarlet fever, or diphtheria.

Probably the best of all guides to the nature and severity of the infection is the temperature. An excessively high temperature usually indicates a severe type of infection. Some idea of this may be gained from these figures, giving the highest temperature and the mortality in two hundred and thirty-one cases, not including cases with measles or diphtheria:

| HIGHEST TEMPERATURE.    | Cases. | Deaths. | Percentage mortality. |
|-------------------------|--------|---------|-----------------------|
| 106° F. or over .....   | 55     | 47      | 85.5                  |
| 105° or 105.5° F. ....  | 94     | 56      | 60.0                  |
| 104° or 104.5° F. ....  | 53     | 26      | 49.0                  |
| 102° to 103.5° F. ....  | 22     | 13      | 60.0                  |
| 99.5° to 101.5° F. .... | 7      | 5       | 71.0                  |

The high mortality of the cases with unusually low temperature is due to the fact that they nearly always were seen in infants with very feeble vitality. The outlook in cases with a steadily high temperature—between 102.5° and 104° F.—is usually more favourable than in those with wide fluctuations, such as 100° to 105.5° F. As a rule, the danger from the disease increases steadily with every degree of temperature above 104.5° F.

An important factor in the prognosis is the previous condition of the patient. The association with rickets is unfavourable, both on account of the feeble muscular power of these children and their thoracic deformities. Marked and persistent tympanites is always an unfavourable symptom. Any condition which diminishes the general vitality increases the danger from broncho-pneumonia. As a rule, second attacks are more serious than the primary ones, especially if the interval between them is short.

In making the prognosis in any given case, the symptoms to be con-

sidered are the height and course of the temperature, the presence or absence of nervous symptoms, the condition of the organs of digestion, the presence of cyanosis and the extent of the disease as shown by the physical signs. I have not found the examination of the blood to aid much in prognosis.

Nervous symptoms early in the disease do not affect the prognosis. Three cases in which convulsions occurred at the onset recovered, but of thirty-seven cases in which convulsions occurred at a late period during the course of the disease, all but one proved fatal.

So long as the nutrition of the patient can be well maintained, no protracted case is hopeless, no matter how extensive the local disease may be; but the existence of vomiting, diarrhoea, or persistent tympanites makes the issue doubtful, even though the other symptoms are favourable.

**Treatment.**—The most important part of prophylaxis is to give careful and early attention to every attack of bronchitis in an infant, for every such attack should be regarded as a possible precursor of pneumonia. It is striking that one sees broncho-pneumonia so seldom in private practice among the better classes, even though bronchitis is very frequent; while among hospital and dispensary patients, where bronchitis is very often neglected, broncho-pneumonia is constantly seen. Cases of measles and diphtheria which are complicated by pneumonia should, if possible, be carefully isolated from others, and wards in which they are treated should be thoroughly disinfected before they are used for simple cases.

The hygienic treatment of broncho-pneumonia is important, and usually it receives too little attention. It is much the same as that of cases of acute bronchitis already discussed. What was said in that connection regarding the necessity for fresh air and the caution as to very cold air, may be here repeated. The cold-air treatment is not admissible in very young or delicate infants, nor in cases of disseminated pneumonia (capillary bronchitis). The best results from this treatment are seen in the cases with extensive consolidation and with the minimum amount of bronchitis, and it is to be highly recommended in the pneumonia of the severe acute infections—diphtheria, measles, and scarlet fever. The dress and protection of the patient with the cold-air treatment are discussed under Lobar Pneumonia.

Older children with pneumonia should be kept in bed. Infants for a considerable part of the time may be held in the nurse's arms. A frequent change of position in all cases is essential; no child should be allowed to lie for hours directly on the back. The general rules previously laid down for feeding all sick children should be followed here. As a rule, medicine should not be administered in the food.

The same local treatment may be employed as in cases of bronchitis.

of treatment should be followed, cod-liver oil, arsenic, and iron being used, according to the indications in each particular case.

One should never declare one of these cases of protracted pneumonia to be hopeless, nor should he be too ready to assume that tuberculosis is present because the child is wasted and anæmic, and the physical signs have persisted.

No specific treatment of pneumonia has yet been proposed which can be recommended.

## CHAPTER V.

### DISEASES OF THE LUNGS.—(Continued.)

#### LOBAR PNEUMONIA.

(*Fibrinous Pneumonia; Croupous Pneumonia.*)

**Etiology.**—*Age.*—Lobar pneumonia may occur at any age. I have seen it in an infant of three months; but it is not until after the first year that it begins to be frequent. After the third year nearly all the cases of primary pneumonia are of this variety.

Of 500 cases the ages were as follows:

| AGE.   | Cases. | Per cent. |
|--|--------|-----------|
| During the first year . . . . .                    | 76     | 15        |
| From the second to the sixth year . . . . .        | 309    | 62        |
| "    "    seventh to the eleventh year . . . . .   | 104    | 21        |
| "    "    twelfth to the fourteenth year . . . . . | 11     | 2         |
| Totals . . . . .                                   | 500    | 100       |

*Season.*—In 136 cases the seasonal occurrence was as follows:

| SEASON.                              | Cases. | Per cent. |
|--------------------------------------|--------|-----------|
| In the three winter months . . . . . | 48     | 35        |
| "    "    spring    "    . . . . .   | 62     | 46        |
| "    "    summer    "    . . . . .   | 6      | 4         |
| "    "    autumn    "    . . . . .   | 20     | 15        |
| Totals . . . . .                     | 136    | 100       |

Lobar pneumonia, in children therefore, as in adults, occurs most frequently during the spring months. April shows the largest number of any single month.

*Previous Condition.*—In my hospital cases, eighty-two per cent of the children were previously in good condition, and only eighteen per cent

were delicate, rachitic, or syphilitic. This observation has been borne out by my experience in private practice, viz., that as a rule lobar pneumonia affects children who were previously healthy. Or to state the matter differently, if a strong child contracts pneumonia it is nearly always of the lobar variety.

*Previous Disease.*—Previous attacks of pneumonia are observed in but a small proportion of cases. It was noted only five times in 160 cases. In the vast majority of cases lobar pneumonia is a primary disease, although it occasionally occurs as a complication of pertussis, measles, typhoid or scarlet fever, and even diphtheria—chiefly, however, in children over three years old.

Epidemics of lobar pneumonia I have never witnessed, although on several occasions I have seen two children in a family attacked either simultaneously or in rapid succession. Exhaustion, fatigue, and exposure are to be ranked as associated exciting causes.

In addition to other causes, there is required for the production of the disease the presence and growth of the pneumococcus. Associated with it are often found the staphylococcus aureus and occasionally the bacillus of influenza.

*Lesions.—The Seat of the Disease.*—In 950 cases in children under fourteen years, this was as follows:

| SEAT OF DISEASE.                      | Personal cases. | Collected cases. | Totals. |
|---------------------------------------|-----------------|------------------|---------|
| Right lung, upper lobe only . . . . . | 39              | 137              | 176     |
| “ “ middle “ “ . . . . .              | 8               | 4                | 12      |
| “ “ lower “ “ . . . . .               | 26              | 142              | 168     |
| “ “ more than one lobe . . . . .      | 13              | 64               | 77      |
| Totals, right lung . . . . .          | 86              | 347              | 433     |
| Left lung, upper lobe only . . . . .  | 25              | 68               | 93      |
| “ “ lower “ “ . . . . .               | 49              | 214              | 263     |
| “ “ more than one lobe . . . . .      | 9               | 29               | 38      |
| Totals, left lung . . . . .           | 83              | 311              | 394     |
| Both lungs, upper lobes . . . . .     | ..              | 13               | 13      |
| “ “ lower “ “ . . . . .               | 3               | 38               | 41      |
| “ “ elsewhere . . . . .               | 9               | 60               | 69      |
| Totals, both lungs . . . . .          | 12              | 111              | 123     |

The right lung was thus affected in 45.5 per cent; the left lung in 41.5 per cent; both lungs in 13 per cent. In the order of frequency, the disease involves, first, the left base; second, the right apex; third, the right base; forth, the left apex. The disease affects, as a rule, a single lobe, and often only a circumscribed portion of a lobe.

Lobar pneumonia among children is so rarely fatal that the opportunities for a study of the peculiarities of the lesion have been somewhat limited. The anatomical changes resemble those seen in the adult lung. There is an exudation into the alveoli and smaller bronchi of fibrin, serum, leucocytes, and red blood-cells (Fig. 71). There is usually in addition an inflammation of the mucous membrane of the larger bronchi and of the pleura. The frequency and severity of the pleurisy is a peculiarity of the lesion in children.

In the first stage, that of *congestion*, the portion of lung involved is dark-coloured, heavy, and œdematous, and shows under the microscope a serous and cellular exudation into the air vesicles, with swelling of the epithelial cells lining the alveoli.

In the second stage, that of *red hepatisation*, there is usually some exudation upon the pulmonary pleura, generally a thin layer of fibrin, giving it a dull, granular look. The lung itself is of a uniform dark-red colour. It is solid and cuts like liver. It looks as if it had been inflated to its utmost extent and then injected with a material which had solidified. The consolidated area is sharply defined. Under the microscope the air vesicles are seen to be distended with an exudation which is chiefly fibrin, but with some leucocytes, red blood-cells, and desquamated epithelial cells. The cells are chiefly leucocytes, and are usually more abundant than in the pneumonia of adults.

In the third stage, that of *gray hepatisation*, the lung is more moist, and the inflammatory products are partly decolourised. This change takes place irregularly throughout the lung, giving it a mottled appearance.

The fourth stage, that of *resolution*, follows gray hepatisation, and consists in the degeneration and liquefaction of the products of inflammation, which are ultimately carried away by the lymphatics, or pushed out into the bronchi and removed by coughing.

The duration of the stage of congestion is from a few hours to several days; that of the stage of red hepatisation from two days to two or three weeks. This is the condition in which the lung is most often seen at autopsy. The stage of gray hepatisation is commonly shorter. Resolution usually begins when the temperature falls to normal, but occasionally it may be delayed for several days. It is generally complete in about a week.

*Variations in the Lesions.*—(1) Instead of clearing up at the usual time, the lung may remain consolidated for several weeks, and then resolve. (2) The stage of gray hepatisation may be followed by a great exudation of pus cells, which may everywhere infiltrate the affected lung; or these may be circumscribed so as to form a single large abscess or many small ones. (3) There may be small areas of gangrene. All these conditions are very rare in children. (4) There may be excessive pleu-

ris, or pleuro-pneumonia. This is found at autopsy in about one-half the cases, and will be separately considered elsewhere.

The lesions in the other organs are for the most part due to the pneumococcus. There may be pericarditis, especially with pneumonia of the left side, if complicated by excessive pleurisy. This is seen even in infants. The pericardial inflammation closely resembles that of the pleura. There is a very abundant exudation of fibrin and pus, coating both surfaces of the pericardium. Acute meningitis is rather rare. It is an acute purulent inflammation, with a very abundant exudation of greenish-yellow fibrin and pus, chiefly at the convexity. Less frequently peritonitis is present. Acute parotitis and acute arthritis are seen as rare complications of pneumonia. In most of the complicated cases the other lesions are second to those in the lungs; but they may begin simultaneously with, or even precede, the pneumonia. In cases with complications other than thoracic ones, a general pneumococcus septicæmia is usually present. From reports thus far published it would appear that pneumococci are found in the blood of children with pneumonia much less frequently than in that of adults. In seventy cases examined by Otten, positive blood cultures were obtained but nine times; while in adults fully half the cases give positive results.

The heart is generally found in diastole, with the cavities, especially those of the right side, distended with soft clots. There may be found ante-mortem thrombi, which may extend into the pulmonary artery or the aorta.

**Symptoms.**—(1) *The Typical Course.*—A child three or four years of age, after a few hours of slight indisposition, is suddenly taken with vomiting, followed by a rapid rise in temperature. He is dull and heavy, complains of headache and general weakness, refuses food, and is easily persuaded to remain in bed. He has the appearance of being quite ill, even after a few hours. Occasionally sharp pain in the side is complained of. The skin is dry; there are marked thirst, restlessness, and the other symptoms which accompany fever. The temperature is found to be 104° F., or even higher; the respirations 40 to 50 a minute; the pulse full, strong, and 120 to 130. On the second day the patient is no better. The temperature remains high; the tongue is coated; the anorexia continues; the pain is more severe; cough is present and may be quite frequent.

After the second or third day the patient is usually more comfortable, and sleeps better, but may be disturbed by the cough. At times there is restlessness, and at night there may even be slight delirium. The respiration continues rapid and the temperature high. These general symptoms show very little change until the sixth or seventh day, when, after a long sleep, which has been more natural than before, the patient wakes, decidedly improved as to all his symptoms. There is less fever,



and the temperature continues to fall rapidly until it touches the normal line, or it may even go below this. As the fever subsides the pulse drops to 90 or 100, and the respirations to 25 or 30 a minute. The appetite soon returns, and convalescence is usually rapid. In a week the patient is out of bed, and in two or three weeks more he is out of doors. This is the course seen in fully two-thirds of all the cases of lobar pneumonia at this age.

(2) *Pneumonia of Short Duration*.—Instead of running the usual course of from five to eight days, cases are seen in which the duration is only three or four days, although the physical signs indicate that the process in the lung passes through the usual stages. These differ from the ordinary type chiefly in their duration. They are always mild.

(3) *Abortive Pneumonia*.—This form of the disease is rarely seen in hospitals, but it is not infrequent in private practice where the physician is summoned at the earliest signs of illness. The onset is precisely like that of ordinary pneumonia, and may even be as severe as the average case. The physical examination of the chest gives all the signs of the first stage of the disease, but on the second or third day the physician is greatly surprised to find that the temperature has fallen to normal, and that all the physical signs have disappeared. The process in such cases does not seem to go beyond the first stage of congestion; there is no evidence of hepatisation of the lung. The course is often such as to lead the physician to the opinion that he has made a mistake in his diagnosis. This type of pneumonia corresponds with abortive types of other infectious diseases so frequently met with in children. The temperature curve in such a case is shown in Fig. 89. The diagnosis of these cases is always attended with some uncertainty. There can be no doubt that many of the unexplained high temperatures of brief duration which are seen in children are from this cause. Exactly why it is that the disease sometimes terminates in this way can not always be explained. It may be because the resistance of the patient is greater than usual, or the virulence of the pneumococcus is less.

(4) *The Prolonged Course*.—Although usually lasting about a week, it is not rare for pneumonia to continue ten, twelve, or even fifteen days. This prolonged course is usually due to the fact that the disease spreads from one part of the lung to another, or even to the opposite lung, involving in succession two, three, or more lobes. This is sometimes known as "creeping" pneumonia; it is always severe and the outlook is generally unfavourable. A prolonged temperature with physical signs limited to a single lobe should always suggest complications, most frequently empyema, occasionally pericarditis.

(5) *Cerebral Pneumonia*.—This term was first applied by Rilliet and Barthéz to cases of pneumonia in which the cerebral symptoms predominate. They will be considered later.

*Onset.*—Prodromal symptoms of more than a few hours' duration are quite rare. The onset of lobar pneumonia is almost invariably abrupt, with well-marked symptoms—vomiting, diarrhœa, chill, or convulsions. Vomiting is altogether the most frequently seen. In summer particularly, there may be vomiting and diarrhœa. A distinct chill is rare in a child under five years of age, and is not very common even in older children. Convulsions are not very infrequent, being seen in about five per cent of the cases. Their occurrence depends upon the suddenness of the invasion and the susceptibility of the patient.

*Cough.*—This is present in most of the cases throughout the disease, but often is not marked for the first day or two. It is seldom a distressing symptom. A disposition to suppress the cough on account of pain is very frequently noticed.

*Expectoration.*—This is rarely seen in early childhood, and practically never under five years of age. Children of ten or twelve may have the same expectoration as adults—white and viscid, or brownish-red early in the disease, yellow and abundant toward its close. This shows the presence of the pneumococcus in great numbers.

*Pain.*—Headache and general muscular pains in the back and extremities are frequent during the invasion. The characteristic pain, however, is pleuritic. It is not necessarily felt in the region of the affected lung, and often not in the chest at all. It is frequently referred to the loin, the epigastrium, or to any region to which the intercostal nerves are distributed. I have seen a number of cases in which there was intense localised pain in the right iliac fossa, associated with such extreme tenderness as to lead to the suspicion that the case was one of appendicitis.

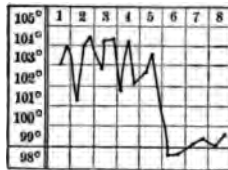
*Prostration.*—This is one of the characteristic features of pneumonia. The patient is generally willing to go to bed on the first day of the attack, and shows little desire to leave it while the disease continues. "Walking cases" are not common in children.

*Respiration.*—This is always accelerated, and generally out of proportion to the pulse. The normal ratio of the respiration to the pulse is one to four; in pneumonia, frequently one to two. The respiration is not laboured and not quite panting, although this term is sometimes used to describe it. It is jerky. There is a short inspiration, then a momentary pause, followed by a quick expiration, which is accompanied by a short moan. This expiratory moan is very characteristic. The rapidity of respiration is usually in proportion to the amount of lung involved, but it is also modified by the temperature, as the respirations often drop from 60 to 30 in the course of a few hours at the crisis.

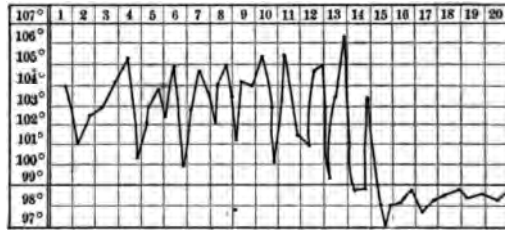
*Pulse.*—In the early part of the disease this is frequent, full, and strong, from 120 to 150 a minute. Later it may be weak, small, compressible, and sometimes irregular. It is much more rapid in the child

than in the adult, 160 and 180 being often seen in cases not especially severe. The pulse rate is of less importance than its character.

*Temperature.*—The typical temperature curve of lobar pneumonia (Fig. 86) is characterised by an abrupt rise usually to 104° or 105° F., and by daily fluctuations generally within the limits of two or three

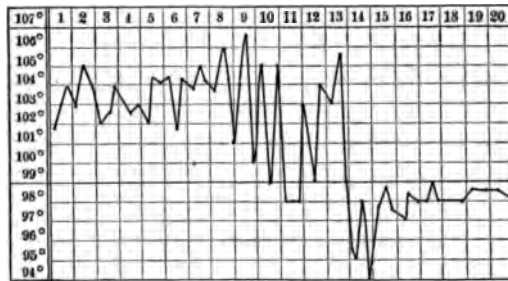


**FIG. 86.—TYPICAL TEMPERATURE CURVE OF LOBAR PNEUMONIA.** *History.*—Male, three years old; in fair condition; sudden onset; signs of consolidation—bronchial respiration and voice, and dullness—over left lower lobe behind, not distinct until the morning of the fifth day. On the seventh day the lung was resolving.



**FIG. 87.—LOBAR PNEUMONIA WITH REMITTENT TEMPERATURE.** *History.*—Female, eighteen months old; in fair condition; sudden onset; repeated examinations of chest made, but no abnormal signs until the ninth day, when there were very rude respiration and slight dullness at the right apex, in front; on the twelfth day all the signs of consolidation at the same point, no râles; four days after the crisis the lungs were clear.

degrees until the crisis, at which time the temperature falls to normal, usually in the course of twenty-four hours. After this time it does not go above the normal line. Such a curve is seen in the majority of cases



**FIG. 88.—LOBAR PNEUMONIA WITH SUBNORMAL TEMPERATURE AFTER THE CRISIS.** *History.*—Female, nineteen months old; fairly healthy; sudden onset; symptoms typical but physical signs delayed; consolidation in left mammary region on the eighth day; on the ninth in right lung middle lobe; on the eleventh day a pseudo-critical drop followed after twenty-four hours of apyrexia by a further rise, which was accompanied by signs of extension of the disease in the right lung. Resolution rapid after crisis.

over three years of age.

In cases under three years of age it is not uncommon for the temperature to be of a more or less remittent type (Fig. 87).

These wide fluctuations often lead to great difficulty in diagnosis, particularly if the physical signs appear late, as they not infrequently do. It is probable that some of them are to be explained as mixed infections.

The accompanying chart (Fig. 88) illustrates three features which are often seen in pneumonia: (1) A temperature which early in the disease is steadily high and as

the day of crisis approaches becomes remittent; (2) a secondary rise after being normal for twenty-four hours, which was due in this instance to an extension of the disease to a new part of the lung; (3) a fall to a point considerably below normal at the time of the crisis. In this case the temperature fell in the course of eighteen hours from 105° to 95° F., and later still lower; it was two days before it finally remained at the normal point. A fall to 96.5° or 97° F. at the time of crisis is not uncommon.

In the foregoing cases the fever terminated by crisis. In Fig. 89 is shown one ending by lysis. This is a mode of termination much more frequent in young children than in those who are older. Thus, in 93 of my own cases, nearly all of which were under three years of age, the fever ended by crisis in 49, and by lysis in 44; while in 552 collected cases, the majority of which were in older children, 396 ended by crisis, and 126 by lysis.

The following table shows the day of crisis in 567 cases of lobar pneumonia in children who recovered:

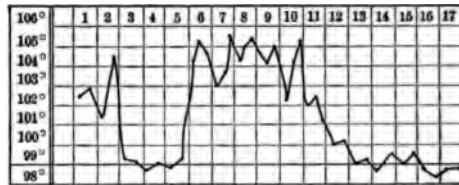


FIG. 89.—<sup>\*</sup>ABORTIVE PNEUMONIA IN LEFT LUNG, FOLLOWED BY TYPICAL PNEUMONIA IN RIGHT LUNG, TERMINATING BY LYSIS. *History.*—Male, seventeen months old, healthy; sudden onset; on the second day disseminated fine râles in both lungs behind, and over left lower lobe very feeble respiration, high-pitched—i. e., some bronchitis, with congestion (?) of left base. On the third, fourth, and fifth days, general symptoms gone and signs nearly disappeared. On the sixth day all symptoms of pneumonia, and on the seventh distinct consolidation of right base, rest of chest clear. Subsequent course typical; resolution rapid and complete.

#### *The Day of Crisis.*

|                      |          |                            |           |
|----------------------|----------|----------------------------|-----------|
| Second day . . . . . | 3 cases. | Eleventh day . . . . .     | 18 cases. |
| Third " . . . . .    | 22 "     | Twelfth " . . . . .        | 7 "       |
| Fourth " . . . . .   | 43 "     | Thirteenth day . . . . .   | 8 "       |
| Fifth " . . . . .    | 88 "     | Fourteenth " . . . . .     | 7 "       |
| Sixth " . . . . .    | 83 "     | Fifteenth " . . . . .      | 1 case.   |
| Seventh " . . . . .  | 132 "    | Eighteenth " . . . . .     | 3 cases.  |
| Eighth " . . . . .   | 73 "     | Twenty-first day . . . . . | 1 case.   |
| Ninth " . . . . .    | 55 "     | Twenty-sixth " . . . . .   | 1 "       |
| Tenth " . . . . .    | 22 "     |                            |           |

567

From this table it will be seen that the most frequent critical day is the seventh, and that in sixty-six per cent of the cases it was from the fifth to the eighth day. The causes of a post-critical rise in the temperature are chiefly two—extension of the disease to a new area, or the development of pleurisy, which is apt to be purulent. Less frequently it is due to otitis, meningitis, pericarditis, or gastro-enteritis. In fatal cases

the temperature is generally high until the end. In general, it may be said that the temperature is considerably higher in children than in adults; in the majority of cases it reaches 105° F., the usual range being from 102° to 105° F. In 15 of 137 cases, or eleven per cent, it reached 106° F. or over.

*Gastro-enteric Symptoms.*—These are more common in infants than in older children. At the onset there is frequently vomiting, sometimes also diarrhœa. A continuance of the vomiting is rare, and is generally due to improper feeding or medication. It may be a very serious complication. Diarrhœa is also rare, except at the onset and in summer cases. Great tympanites is a distressing symptom, and when present, it is a bad prognostic sign. Throughout the disease there are anorexia, coated tongue, and the usual symptoms of high fever.

*Nervous Symptoms.*—Cerebral symptoms are frequent and very often misleading. Pneumonia is often ushered in by convulsions, which may be repeated two or three times in the course of the first twenty-four hours. They are sometimes followed by drowsiness or stupor, sometimes by active delirium. Cerebral symptoms may predominate for several days. There may be opisthotonus, dilated or contracted pupils, irregular pulse, retracted abdomen, and, in fact, almost every symptom of meningitis. Lumbar puncture in these cases usually shows an excess of cerebro-spinal fluid under high tension and it may contain a few pneumococci. Occasionally the decubitus *en chien de fusil*, or gun-hammer position, is assumed. These are often described as cases of *cerebral pneumonia*, and in many of them pneumonia is not suspected until the fourth or fifth day of the disease, sometimes not until the crisis occurs, when the rapid disappearance of all these nervous symptoms indicates their origin. Early convulsions are not generally followed by an especially severe type of the disease, only one of seven such cases proving fatal. On the other hand, cases with late convulsions are usually fatal, as they indicate either a very severe form of the disease or the development of a serious complication, usually meningitis.

Delirium is much more frequent than convulsions, and is seen in nearly one-fourth of the cases. Generally it is slight and noticed only at night or when the temperature is very high. It is most pronounced at the height of the disease. Other nervous symptoms belonging to the typhoid state are occasionally seen, but only in the worst forms of the disease.

I have been unable to discover any relation between the seat of the disease in the lungs and the occurrence of cerebral symptoms. They are more frequent in children under five years than in those who are older, and depend upon the suddenness of the invasion, the intensity of the infection, and the susceptibility of the child. Late in

the disease they may indicate exhaustion, toxæmia, or complicating meningitis. The usual nervous symptoms—restlessness, headache, sleeplessness, etc.—are nearly always proportionate to the height of the temperature.

*Urine.*—Throughout the febrile period of the disease the urine is scanty, high-coloured, with a high specific gravity, usually loaded with urates and with marked diminution of the chlorides. In a small number of cases a trace of albumin may be found, and occasionally a few hyaline casts. Evidences of serious renal disease I have seldom found in lobar pneumonia, and in the experience of all observers it is extremely rare in early life.

*Skin.*—The face, in pneumonia, is usually flushed, sometimes on both sides and sometimes only on one; in other cases it is pale, but not indicative of pain. Cyanosis is rare except toward the close of the disease and is usually a sign of respiratory failure. Herpes of the lips or face is quite frequent.

*Blood.*—There is regularly a leucocytosis of from 20,000 to 50,000; the increase is chiefly in the polymorphonuclear cells which usually form from seventy to eighty-five per cent of the leucocytes. (See also chapter on Diseases of the Blood.)

**Physical Signs.**—The earliest signs in pneumonia are due to the acute congestion of the affected lung or lobe, in consequence of which less air enters this portion and more air the rest of the lungs. Percussion gives diminished resonance or slight dulness, often of a somewhat tympanitic character over the affected area, and exaggerated resonance over the remainder of this lung and over the opposite lung. Auscultation over the affected lobe gives feeble respiratory murmur, rather high in pitch; sometimes there may be absence of all breath-sounds so complete as to suggest fluid. The normal respiratory murmur over the healthy portions of the lungs is intensified. In children this exaggerated breathing is not infrequently mistaken for bronchial breathing, and the physician may be led into the error of locating the pneumonia upon the wrong side. Exaggerated breathing does not differ from normal breathing except in intensity. Bronchial breathing is higher in pitch, tubular in character, and is heard with nearly equal intensity, both on expiration and inspiration. If the chest is frequently auscultated, crepitant or fine subcrepitant râles (Figs. 90 and 91) may usually be heard at some period at the end of full inspiration, but often they are present but for a few hours, and they may be missed altogether.

In the second stage, that of consolidation (Fig. 92), no air enters the air vesicles of the affected portion of the lung. Upon palpation there is found here exaggerated vocal fremitus, and on percussion there is marked dulness, but very rarely flatness. Over the rest of this lung



**FIG. 90.—FIRST STAGE.** Congestion of left lower lobe, with crepitant râles. Feeble breathing of a rude character, with slight dulness.



**FIG. 91.—**In the centre of the area, a small spot of pure bronchial breathing and voice; surrounding this an occasional crepitant râle, with broncho-vesicular breathing and slight dulness.



**FIG. 92.—SECOND STAGE.** Complete consolidation of left lower lobe. Pure bronchial breathing and bronchial voice; marked dulness; increased vocal fremitus, and at the lower part a few friction sounds.

**NOTE.**—During resolution the signs take the inverse order: those of Fig. 92 give place to those of Fig. 91, and these in turn to those of Fig. 90. In addition, many coarse râles may be heard.

there is exaggerated, sometimes even tympanitic, resonance; this is especially frequent at the apex of the lung in front, when there is consolidation at the base behind. Under these conditions cracked-pot resonance may sometimes be obtained. Over the healthy lung there is exaggerated resonance. On auscultation over the consolidated portion there are bronchial breathing and bronchial voice, the area over which they are heard being sharply defined. Râles are usually absent, but there may be pleuritic friction sounds.

In the stage of resolution there is a gradual disappearance of the signs of consolidation. The pure bronchial is replaced by broncho-vesicular breathing, the vesicular element gradually predominating. Moist râles of all varieties are heard. Usually the most persistent signs are slight dulness or diminished resonance, with a respiratory murmur which is feebler than normal and a little higher in pitch; sometimes there are also dry friction sounds. These signs may persist for two or three weeks.

*Exceptional Physical Signs.*—While in the majority of cases the signs of consolidation are distinct on or before the fourth day, in not a few they may be delayed much longer. Of eighty-two cases in which the day was noted on which consolidation was found, it was not until the fifth day or later in one-fourth the number. In six of them, although carefully and repeatedly examined, no consolidation was found until the seventh day or later and in one case not until the twelfth day. It has been customary to look upon these cases of delayed or concealed physical signs as cases of central pneumonia. That pneumonia may exist in the centre of a lung for a number of days is, to my mind, extremely improbable. At autopsy, superficial pneumonia I have very frequently seen, but central pneumonia never. There are two regions in which pneumonia may exist and yet not be accessible by our means of physical examination, viz., at the apex of the lung in the part covered by the shoulder, and along the posterior border of the lung where it lies against the vertebræ. In either of these situations pneumonia may be present without our being able to find it. It is quite common in cases with late physical signs that the first distinctive evidences of disease are found high in the axilla, or beneath the clavicle in front, and these regions should be closely watched in doubtful cases. Sometimes the delay is best explained by assuming that constitutional symptoms due to a pneumococcus infection may be present for several days before the development of the local lesion in the lung.

*Complications.*—The occurrence of dry pleurisy over the consolidated portion of the lung is so constant that it can hardly be considered a complication. A slight serous exudation of two or three ounces is not uncommon, but more than this is rare in young children. In the most severe cases of pleurisy there is an excessive exudation of fibrin and pus.



This occurred in eight per cent of my cases. This variety is known clinically as pleuro-pneumonia, and will be considered separately. Pericarditis is uncommon. It is seen more often in infants than in older children. It most frequently develops at the height of the pneumonia and occurs rather oftener when this affects the left lung than the right; it occurs in pleuro-pneumonia more often than in the simple form. The pericarditis is usually of the fibrino-purulent type. It may sometimes be discovered by physical signs; but rarely gives rise to any new symptoms. Endocarditis was not seen in my cases, though it occasionally occurs. Meningitis is rare, and generally develops late in the disease. It is nearly always ushered in by repeated attacks of vomiting or convulsions. Its course is short and progressive. Peritonitis causes few new symptoms except abdominal distention, pain, and tenderness. Parotitis and arthritis are very rare and are easily recognised.

**Course and Termination.**—In the great majority of cases lobar pneumonia terminates either in perfect recovery or in death. When ending in recovery, resolution commonly begins immediately upon the cessation of the fever, and is complete in about a week. Delayed resolution is not common in children; chronic pneumonia and tuberculosis are rare sequelæ, but empyema is very frequent. Its symptoms sometimes develop immediately after the pneumonia, the temperature continuing high; or there may be an interval of a few days before the development of the pleural symptoms. Some pleuritic adhesions probably remain in every case in which there has been much dry pleurisy, and when severe and extensive, these may be the cause of subsequent symptoms, like any other dry pleurisy.

Death from uncomplicated pneumonia may be due to exhaustion, or to heart failure, with or without failure of the respiration. The signs of heart failure sometimes develop quite rapidly in cases which are apparently doing well. The symptoms are: coldness of the hands and feet, then of the legs and arms; a rapid, compressible, and sometimes irregular pulse; muscular weakness and pallor, but usually no cyanosis. The symptoms of respiratory failure are: very rapid superficial respirations, sometimes 100 a minute; blueness of the lips and finger nails; often a leaden hue of the whole body; there are loud tracheal râles, and recession of all the soft parts of the chest on inspiration.

Death may occur early in the disease, where the pneumonia has spread rapidly, involving both lungs. In most of the uncomplicated fatal cases, death results from failure of the circulation at about the end of the first week. In the complicated cases death usually occurs in the second week; but I once knew fatal meningitis to develop at the end of the fourth week.

**Diagnosis.**—The most characteristic differences between broncho- and lobar pneumonia are shown in the following table:

## BRONCHO-PNEUMONIA.

1. Often secondary.
2. Under two, chiefly under one year.
3. Occurs more frequently in delicate and debilitated children.
4. Bacteria—in primary cases, usually the pneumococcus; in secondary cases, usually mixed infection.
5. Products of inflammation chiefly cellular; process often diffuse.
6. Onset often gradual, sometimes insidious, especially when secondary.
7. No typical course; fever often lasts three or four weeks; rarely terminates by crisis.
8. Involves both lungs as a rule, most frequently lower lobes posteriorly.
9. Signs of bronchitis mingled with those of consolidation; râles in other parts of the same lung, or in the opposite lung, throughout the disease.
10. Consolidation later—fourth to seventh day: there may be none; apt to be incomplete; shades off gradually.
11. Resolution slow, one week to two months; often incomplete; strong tendency to become chronic.
12. Relapses and second attacks frequent.
13. Sequelæ: Empyema, chronic interstitial pneumonia, sometimes tuberculosis.
14. Prognosis always serious from the age and the circumstances under which disease occurs.
15. Hospital mortality 50 per cent of primary cases, 65 per cent of all cases.

## LOBAR PNEUMONIA.

1. Almost always primary.
2. Most common between three and eight years.
3. More often in those previously healthy.
4. The pneumococcus, very often alone.
5. Chiefly fibrin; process circumscribed.
6. Onset sudden, with well-marked symptoms.
7. Typical course; crisis usually from fifth to eighth day.
8. Usually one lobe or a part of a lobe; left base most frequently, right apex next.
9. Râles only early, and during resolution; frequently no signs in opposite lung.
10. Consolidation earlier; second or third day. Consolidation complete; area usually sharply defined.
11. Resolution rapid, usually complete within a week.
12. Both are rare.
13. No sequelæ except empyema.
14. Prognosis good; rarely fatal except from complications — empyema, meningitis, pericarditis.
15. Mortality 4 per cent of all cases.

In the majority of cases the symptoms are plain and the physical signs so typical that it is difficult to overlook pneumonia if any degree of care is used in the examination of the patient. The difficulties in diagnosis are due to the great variation in the general symptoms, and to the late appearance of the physical signs. The error usually made is to mistake pneumonia for some other disease, rather than to mistake some other disease for pneumonia. On account of its frequency in children, pneumonia should always be excluded before accepting any other explanation of a continuously high temperature. The rule should be followed, in all cases of acute illness, of making a thorough examination of the chest daily until the diagnosis is clear. If, to high temperature,

rapid respiration and marked leucocytosis are added, one should always suspect pneumonia, no matter what the other symptoms may be. It not infrequently happens that the general symptoms are quite characteristic and yet the physical signs appear late. In such cases pneumonia should always be looked for high in the axilla or just beneath the clavicle, since it is particularly in the cases of apex pneumonia that this obscurity is likely to exist.

In their onset, scarlet fever, tonsillitis, and gastro-enteritis may all resemble pneumonia. Scarlet fever is recognised by the sore throat and the characteristic eruption on the second day; tonsillitis, by the local symptoms. In infancy, pneumonia often begins with vomiting and sometimes there is also diarrhoea, which may lead one to mistake the disease for gastro-enteritis. The constitutional symptoms of influenza often closely resemble those of pneumonia; the diagnosis is frequently in doubt for several days until definite physical signs of pneumonia make their appearance. From all other general diseases, pneumonia is to be differentiated by the physical signs.

Pneumonia with marked cerebral symptoms sometimes resembles cerebro-spinal meningitis. In both we may have the abrupt onset, convulsions, delirium or stupor, opisthotonus, prostration, and marked leucocytosis. The only positive means of differential diagnosis are by the physical signs in pneumonia, and the findings of lumbar puncture in cerebro-spinal meningitis.

The question sometimes arises in pneumonia with cerebral symptoms, whether or not pneumococcus meningitis also exists. If the nervous symptoms are present from the beginning, there is probably no meningitis. If they develop suddenly during the course or toward the close of the disease, meningitis should be suspected. The only positive means of differentiation is by lumbar puncture.

Lobar pneumonia is to be differentiated from a pleuritic effusion. The most common mistake is to confound empyema with unresolved pneumonia. In pneumonia rarely if ever do the signs point to involvement of an entire lung. There is increased vocal fremitus, dullness, bronchial voice and breathing, and occasional râles or friction sounds. In empyema the whole lung is often affected, there is displacement of the heart, flatness on percussion, diminished or absent vocal fremitus, and although bronchial voice and breathing are present, they are usually distant and feeble. There are no râles or friction sounds. In doubtful cases an exploratory puncture should always be made. Serous effusions give the same physical signs as empyema.

**Prognosis.**—There is probably no disease in which the patient appears so ill, and yet so often recovers completely, as in lobar pneumonia in a child over three years old. Of 1,295 collected cases, chiefly from hospital practice, there were but 39 deaths, a mortality of three per cent.

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In 187 cases of my own there were 21 deaths, a mortality of eleven per cent. Only one of the fatal cases was over two years old. The difference between the mortality among my cases and the general mortality given, is due to the fact that a large proportion of the first group were observed in children under two years, while of the collected cases, the vast majority were in older children. Combining the above figures, we have a total of 1,482 cases with 60 deaths, a mortality of four per cent. In nearly all my cases death was due either to complications or to very extensive disease, as when both lungs were involved, or nearly the whole of one lung. In only one case was an uncomplicated pneumonia of a single lobe fatal.

The prognosis depends upon the age of the patient, the intensity of the infection, as shown by the temperature, the nervous symptoms and the pulse, the presence or absence of complications, and the extent of the local disease. These factors are to be taken into consideration rather than any special symptoms. Early convulsions do not materially affect the prognosis. Late convulsions are always very unfavourable.

The occurrence of vomiting, diarrhoea, or marked tympanites late in the disease is always unfavourable.

A temperature range between 102° and 105° F. is the rule, and within these limits the fever does not affect the prognosis. Even very high temperature does not increase the danger from the disease as much as might be expected. Of fifteen cases in which the temperature reached 106° F. or over, all but three recovered; while of six cases in which it was 106.5° or over, only one died. The highest recorded temperature in my cases—107.5° F.—was in a patient who recovered. A transient rise, even though the temperature may go very high, is seldom serious. Much more serious is a fever which remains steadily above 105° F., as in most cases this accompanies either very extensive disease or pleuropneumonia. The continuance of the fever after the tenth day is a bad symptom; for, although the crisis may be postponed until the twelfth day and occur normally, such a prolonged temperature is an indication of a new focus of disease or the development of complications. In a severe attack, the extension of the disease to another lobe after the fifth day is unfavourable. If resolution does not begin soon after the temperature becomes normal, the development of empyema, or some other pulmonary complication, should be apprehended.

**Treatment.**—In the treatment of lobar pneumonia in children, several cardinal facts are to be kept in mind. It is a self-limited disease, having a strong tendency to recovery in the great majority of cases regardless of the treatment adopted. The fatal cases are almost always in children under two years of age; the rare deaths in older ones are usually due to complications. There is no means of treatment by which pneumonia can be aborted or its course shortened. It follows, therefore,

that the indications are, so far as possible, to make the patient comfortable during his illness, to watch for complications, and to treat the individual symptoms as they arise.

In the majority of cases, hygienic treatment is all that is required. The patient should be kept in bed, no matter how mild the attack; he should be disturbed as little as possible. Most children with pneumonia get too much treatment. There seems to be a decided advantage not only in fresh air, but in cold air. Patients in cold rooms sleep better, and cough less, and altogether seem more comfortable than when carefully housed to prevent their "taking cold." Wide-open windows are desirable even though the room temperature is constantly as low as 50° F. The patient should be properly protected by blankets, flannel wrappers, woolen stockings, and at times a hot-water bag at his feet. Food should be given at regular intervals, not oftener than every three hours. It should not be forced when the patient is suffering only from thirst, especially early in the attack, when the appetite is often completely lost. Water should be allowed freely at all times.

These measures, careful nursing, an occasional dose of codeine (gr.  $\frac{1}{16}$  to a child of three years) when the patient is very restless, fretful, or sleepless, an ice-cap to the head, and cold sponging when the temperature makes him uncomfortable, are usually all that is necessary, except to keep a sharp lookout for complications.

Special symptoms may require treatment. When not severe, the nervous symptoms may be controlled by codeine alone or in combination with small doses of phenacetine or the bromides. Sometimes sponging with tepid water is better than drugs. Severe nervous symptoms, such as delirium, stupor, great restlessness with impending convulsions, when associated with high temperature, call for ice to the head, cold sponging, or the cold pack or bath. Pain, if moderate, may be relieved by counter-irritation, by a mustard paste, by dry cups, an ice-bag, or by a hot poultice; if severe, codeine may be used in addition. The cough is rarely severe enough to require treatment. When it is so severe as to prevent sleep, small doses of Dover's powder or codeine should be given. Antipyretic measures are not necessarily called for even if the temperature is very high. Some nervous children are less disturbed by the temperature than by the means used to reduce it. Under such conditions the temperature should be closely watched, but not necessarily interfered with unless other symptoms develop. The nervous symptoms are a better guide than the thermometer to the use of antipyretics. Cold I believe to be the safest and most certain antipyretic we possess. It may be used as a cold sponge bath, the cold pack or an ice-bag to the chest. There is no objection to the bath except the prejudice of the laity. While cold is applied to the trunk the extremities should be closely watched, and heat applied if necessary. The duration of the pack or

bath, and the frequency of their use, will depend upon the individual case. In the majority of cases stimulants are not required. They are called for when the pulse is weak, compressible, and rapid, when the face is pale and the extremities are cold. The same stimulants are to be employed, and in the same way, as in broncho-pneumonia. Circulatory and respiratory stimulants are usually required in larger quantity at the time of and just after the crisis; they are to be used as in broncho-pneumonia.

#### PLEURO-PNEUMONIA.

Under this term are included cases of pneumonia with an excessive amount of pleurisy, the two processes uniting to produce a single clinical type of disease.

In nearly all cases of lobar pneumonia there is a certain amount of inflammation of the pulmonary pleura, and also in those cases of broncho-pneumonia which are accompanied by any marked degree of consolidation. In both of these conditions the pleurisy is usually co-extensive with the consolidation. But in certain cases, in both forms of pneumonia, the amount of pleurisy is excessive, and this so modifies the symptoms and course of the disease as to require for them a separate consideration. In some it appears that the inflammatory process begins almost simultaneously in the lung and in the pleura; while in others the pleurisy follows the pneumonia. These cases are, I believe, almost invariably due to the pneumococcus, although in some there is a mixed infection.

In 398 hospital cases of pneumonia there were 27, or 6.8 per cent, which could be classed as pleuro-pneumonia, the diagnosis being confirmed either by autopsy or operation. Of 190 fatal cases, 12.5 per cent were cases of pleuro-pneumonia. Most of these hospital patients were under three years of age, and the disease is, I think, more frequent at this period than in older children.

**Lesions.**—Of these 27 cases, 17 were classed as broncho-pneumonia and 10 as lobar pneumonia. The left lung was more frequently affected than the right in the proportion of three to two. In most of the cases the pleura covering the entire lung was involved, even though the pneumonia affected but a single lobe, or only a part of a lobe. In nearly half the cases both lungs were involved, but one to a very much less extent than the other. In a small number of cases the pleurisy was limited to the posterior surface of the lung.

In pleuro-pneumonia both the visceral and the parietal pleura are coated with a layer of yellowish-green fibrin, in thick, shaggy masses, causing adhesions of the lung to the chest wall, the diaphragm, and the pericardium (Plate XII). The exudation varies between one-eighth and one-half of an inch in thickness. It can often be stripped from the lung or scraped from the chest wall by the handful. In its meshes small

pockets may form, which contain only a few drops, or sometimes a drachm, of pus, or less frequently serum. This is the condition in which the lung is usually found where death has occurred at the height of the disease. If the process has lasted longer, larger collections of pus may be present. The lung itself shows the usual changes of pneumonia, and if there has been any considerable accumulation of fluid, there are in addition the evidences of compression.

With pleuro-pneumonia of the left side, the pericardium is occasionally involved. This was seen in two of my cases, the lesions closely resembling those of the pleura. In two cases there was also meningitis, and in one peritonitis, the exudation in all cases having the same characteristics.

An inflammation of the intensity described is very often fatal in the acute stage, if the patient is a child under two years old. Occasionally at this age, and very frequently in older children, we see the later stages of the process. The most frequent course is for more and more pus to be poured out from the inflamed pleura until the chest is filled, the case becoming thus one of empyema. Sometimes the fluid is serous instead of purulent, but this is very rare in infancy. Under other circumstances the exudation is partly absorbed, but the greater part becomes organised so as to form a thick jacket of fibrous tissue which binds the lobe or lung to the chest wall, and interferes seriously with its subsequent full expansion. Chronic interstitial pneumonia may follow.

**Symptoms.**—There is little which distinguishes a case of pleuro-pneumonia except the severity of all the constitutional symptoms; the temperature is often higher, the prostration greater, and the patient in every way impresses one as being more seriously ill than with ordinary pneumonia. Sometimes the thoracic pain is more severe and more constant than is usual in pneumonia. The diagnosis, however, is to be made by the physical signs.

In the early stage the pleuritic friction sounds are unusually prominent; after two or three days the signs of consolidation come out clearly in most cases, but still accompanied by loud friction sounds. After the fibrinous exudation is very abundant, the signs are often obscure and confusing, and there may be at no time well-defined signs of consolidation. There is usually a mingling of the signs of consolidation with those of effusion. There is marked dullness, and sometimes flatness. The vocal fremitus is apt to be diminished, and it may be absent. Bronchial voice and breathing are heard, but they are not distinct as in consolidation; they are, however, feeble and distant, as over fluid. There are usually coarse, moist, crackling pleuritic sounds, but these may be absent. The signs may be found over one entire lung, or they may be limited to the posterior region, and even to a single lobe. They resemble those present over fluid, with one exception—viz., the heart is not displaced.

PLATE XII.



ACUTE PLEURO-PNEUMONIA.

The lungs have been separated in front and spread out to show the whole external surface as seen from behind. The left lung, with the exception of a narrow strip along its anterior border, is completely covered with a thick, ragged exudation of fibrin. The left lower lobe was detached: the right lower lobe deeply connected.





If an exploratory puncture is made, nothing is found; occasionally the exploring needle happens to strike one of the small pockets of pus in the meshes of the fibrin, and a few drops of pus are withdrawn. If an incision is made under the supposition that the case is one of empyema, no more pus may be found, the surgeon coming upon the pulmonary adhesions as soon as the chest is opened. There is scarcely any condition in the chest giving signs more puzzling than those just enumerated. They are, however, easily explained by the pathological conditions present.

**Prognosis.**—The prognosis in pleuro-pneumonia is much worse than in simple pneumonia. In infants the outlook is very bad, the majority of cases dying during the acute stage. Very young children may be overwhelmed with the extent and the intensity of the inflammation, and die in four or five days. In children over two years old the most frequent result is for the case to go on to empyema, which with proper treatment usually terminates in recovery. Where there is organisation of the fibrin with the production of extensive adhesions, the ultimate result is often not so favourable as when empyema develops. Convalescence is usually slow, and the patients are liable to exacerbations of pleurisy; they may suffer for years from the partial crippling of one lung.

**Treatment.**—Cases of pleuro-pneumonia are to be managed like the ordinary cases of pneumonia of the severe type. In some, the excessive pain may call for more active counter-irritation and a freer use of opium than in other forms of pneumonia, and the greater prostration may require that stimulants be given earlier and in larger quantities.

#### HYPOSTATIC PNEUMONIA.

This can not often be recognised clinically, but it is very frequently seen upon the post-mortem table. It represents an inflammatory process of a low grade and is seen to some degree in almost every case where an infant has died of chronic disease. It is particularly frequent in those who have died of marasmus. It invariably occupies a strip along the posterior border of both lungs, and usually of both the upper and lower lobes. This is from one to two inches wide, of a uniform dark-red colour, and is sharply outlined. The pleura is not involved, and the remainder of the lung may be normal, congested, or slightly emphysematous. On section, it is seen that the pneumonic area is quite superficial, rarely involving the lung to a greater depth than half an inch. Under the microscope there is found a distention of the small blood-vessels in the affected area, and the air vesicles are filled with many red blood cells, epithelial cells, and a few leucocytes. Between the areas of consolidation are groups of air vesicles which are normal, congested, or collapsed. It is a lobular rather than a broncho-

pneumonia. The lesions in this form of pneumonia are probably the result of venous stasis, owing to the child's recumbent position.

At autopsy the condition may be confounded with atelectasis. Little significance is to be attached to the finding of hypostatic pneumonia at autopsy, and it alone should never be regarded as a sufficient cause of death, although it is perhaps the only lesion present. During life it may give rise to fine moist râles, which are heard along the spine, usually upon both sides; but there is neither dulness nor bronchial breathing.

The treatment is that of the primary disease.

#### CHRONIC BRONCHO-PNEUMONIA—CHRONIC INTERSTITIAL PNEUMONIA—BRONCHIECTASIS.

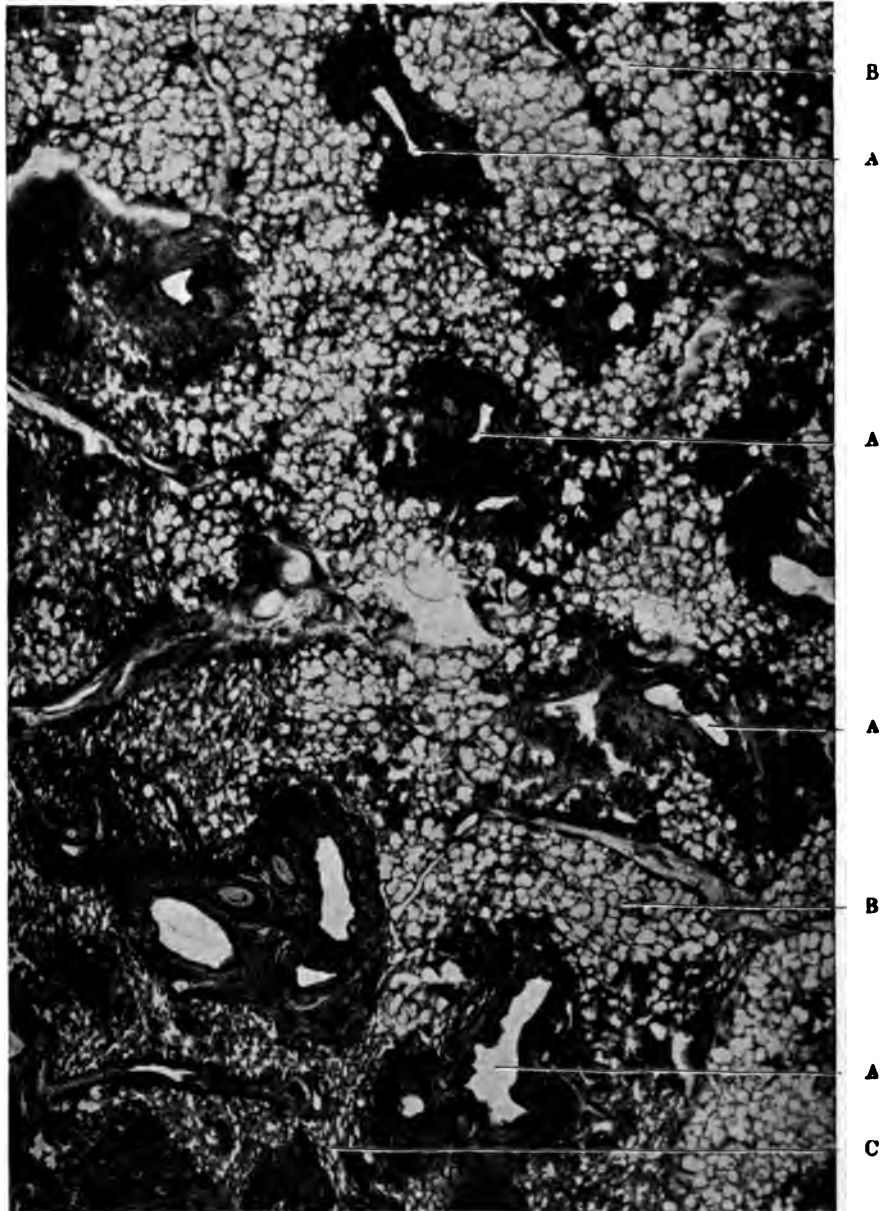
Chronic broncho-pneumonia is an inflammation of the connective-tissue framework of the lung, involving the stroma, the alveolar septa, the walls of the bronchi, and the pleura. It is usually accompanied by cylindrical dilatation of the bronchi—bronchiectasis.

**Etiology.**—In children, as in adults, this process is most frequently associated with pulmonary tuberculosis; but in early life it is not an infrequent condition apart from tuberculosis. The non-tuberculous cases, as a rule, are preceded by an attack of acute broncho-pneumonia, sometimes by several such attacks, separated by longer or shorter intervals.

**Lesions.**—The part of the lung affected may be an entire lobe, but usually it is a portion of one lobe, or there are areas in more than one lobe. There are dense connective-tissue adhesions binding the diseased part to the chest wall, to the diaphragm and to the pericardium, often so firmly that the lung is torn on removal. The affected lung is smaller than in health; it is hard, tough, and fibrous. Surrounding the fibrous portions are emphysematous areas. On section, the process is seen to be somewhat irregularly distributed through the lung, the lesion being usually most marked in the vicinity of the smaller bronchi, and sometimes seen only there, the intervening lung being nearly normal (Plate XIII). In some portions, where the process is most advanced, almost all trace of lung tissue may have disappeared, the part resembling a solid fibrous tumour, through which run the bronchial tubes, usually much dilated. In places this dilatation may be sufficient to form cavities of considerable size. The bronchial glands are often enlarged to the size of a hazelnut, and they may be tuberculous.

Upon examination with the microscope, the pleura is found greatly thickened, with bands of new fibrous tissue passing from it into the lung. The walls of the small bronchi are in most places thicker than normal, but elsewhere they have undergone cylindrical dilatation, and are filled with pus. The walls of the alveoli show a marked proliferation of the

PLATE XIII.



CHRONIC BRONCHO-PNEUMONIA.

In the greater part of the specimen the disease is limited to the vicinity of the small bronchi, A A A, each of which is surrounded by a zone of new connective tissue, the result of the inflammatory process, the intervening lung tissue, B B, being normal. In the lower left-hand portion, the disease is more diffuse; the air vesicles, C, between the areas of new connective tissue are greatly compressed, and in some places entirely obliterated.

(After Delafield.)



connective-tissue elements, and the alveoli are filled with organised inflammatory products, so that they are nearly or quite obliterated. The stroma is much increased in amount throughout the affected lung.

**Symptoms.**—In most of the cases there is a history of an attack of acute broncho-pneumonia, from which the child made a slow convalescence, remaining pale, anæmic, and sometimes wasted for several months. Improvement then took place in the general symptoms, the appetite and strength returned, and in many cases the lost weight was nearly or quite regained. However, neither the pulmonary symptoms nor the physical signs entirely disappeared. There remained a dry, hard cough, which at times was severe. Pains in the chest were occasionally complained of, and perhaps shortness of breath on exertion was noticed.

Examination shows a persistence of the dulness on percussion, with a rude or broncho-vesicular respiratory murmur of very feeble intensity. Little change may take place in these signs for months; then an acute attack of bronchitis or broncho-pneumonia may occur. If the latter, the same lung is affected, and a fresh consolidation is added to the previous disease. This attack may not be very severe, but it drags on for several weeks, with slight fever and little or no change in the physical signs. Partial resolution may then take place, but the lung is left much more seriously crippled than before. Often there is a history of several such attacks, each one leaving the lung a little worse than it found it.

The characteristic physical signs of chronic broncho-pneumonia are not usually present until the process has continued for many months. They may be found over part of a lobe, or over an entire lobe, or even the greater part of one lung. On inspection, there may be seen, in a well-marked case, retraction of the chest, which is especially noticeable when the disease is situated at the apex of the lung. The vocal fremitus is usually increased, but it may not be abnormal. There is marked dulness, often flatness, over the affected area, with exaggerated resonance over the rest of the lung. The area of flatness shades off gradually. The most striking thing on auscultation is the very feeble respiratory murmur; in many cases the lung is almost silent. More rarely there is marked bronchial voice and breathing. Râles and friction sounds are usually absent except during an acute exacerbation of the symptoms, when they may be heard as in any attack of broncho-pneumonia. In recent cases there is no displacement of the heart; in those of long standing it may be drawn far to the affected side by contraction of the adhesions.

When the lesions are once present complete recovery is impossible, and there is always a tendency for them to increase rapidly or slowly, according to the child's vigour of constitution, its surroundings, and the frequency with which exacerbations occur. If the disease is extensive the patient often succumbs to some intercurrent disease or to an acute attack of pneumonia; if limited in area, the process may be arrested and

the patient recover, always, however, to be more or less embarrassed because of the crippling of a part of one lung. Not a small number of these children ultimately die of tuberculosis, and in such cases it is always a difficult matter to decide whether tuberculosis was present from the beginning, or whether it was due to subsequent infection.

The cases in which bronchiectasis is the most important condition are not common. The only characteristic additional symptom is a copious muco-purulent expectoration, which is usually very foetid. It may amount to several ounces a day, and is expelled after paroxysms of coughing, which usually occur in the morning. This may continue for months, or even years, and yet these patients are generally without fever, seldom lose weight, and may have the appearance of being in very good health. It is rare that the physical signs of a cavity are present.

**Prognosis.**—This depends on the extent of the disease, the patient's age and constitution, and on our ability to prevent by treatment, climatic and otherwise, the occurrence of acute exacerbations. Under the most favourable conditions, a few patients may recover completely so far as symptoms are concerned; but the majority remain at best delicate during childhood, or even throughout life.

**Diagnosis.**—The most important thing is to distinguish between the simple and the tuberculous cases, and this, by symptoms and physical signs, is in the majority impossible. If the family history is good, if the patient lives in the country, if his symptoms begin with a well-defined acute attack of pneumonia, if the seat of disease is the base posteriorly, and if the examination of the sputum is negative, the process is probably simple. If the family history is doubtful or is positively tuberculous, if the patient lives in the city, and especially if he is an inmate of an institution or if his home is among the tenements, if the initial symptoms are indefinite, if the disease is situated anteriorly, the process is probably tuberculous. The cutaneous tuberculin test aids much in diagnosis. With a negative reaction tuberculosis can be excluded almost with certainty; but a positive reaction does not prove that the pulmonary process is tuberculous, although it is strongly suggestive. The discovery of tubercle bacilli in the sputum is, of course, conclusive.

Foreign bodies in the lung may give symptoms of chronic bronchopneumonia; metallic and most solid substances may be detected by the X-ray.

**Treatment.**—Nothing has any essential influence upon the disease except change of climate. This should be the same as for tuberculous cases. The treatment of the patient has for its object the maintenance of the general nutrition at its highest point, by careful feeding, judicious exercise, and by most of the measures enumerated in the chapter on Malnutrition. Cod-liver oil should be given throughout every winter season. The cough may be treated as in cases of chronic bronchitis.

Cases of bronchiectasis may obtain considerable relief from inhalations of creosote. They should not be operated upon.

#### ABSCESS OF THE LUNG.

Multiple small abscesses are not uncommon as a termination of acute broncho-pneumonia, in which connection they have already been considered. Larger non-tuberculous abscesses of the lung are rare, very obscure in their symptoms, and apt to be mistaken for localised empyema, sometimes for interstitial pneumonia with bronchiectasis. Three such cases have come under my observation.<sup>1</sup> One was discovered at autopsy, the other two were recognised during life and successfully treated by operation. Other examples in young children have been reported by Huber and by Hedges. The cause of these single abscesses is usually a previous attack of acute primary pneumonia, less frequently an inflammation excited by a foreign body in the lung.

An abscess due to a foreign body is usually accompanied by wasting, and a widely fluctuating temperature of a hectic type—symptoms suggestive of a rapidly advancing tuberculous process. If the abscess follows an ordinary pneumonia the course is generally less intense. The constitutional symptoms differ little from those of empyema. There is an irregular type of fever, sometimes quite high, but more often only from 99° to 101° or 102° F., a moderate cough, not much wasting, and generally not very marked prostration. A leucocytosis of 30,000 to 50,000 is usually present. The physical signs are somewhat confusing and are a combination of those present in effusion and consolidation. There is an area of flatness shading off into dulness. The vocal fremitus may be increased or it may be diminished. The respiratory murmur is very feeble or absent over the abscess, often it is broncho-vesicular in character. Friction sounds and râles are usually present. The heart is slightly or not at all displaced. If an exploratory needle is introduced, pus may not be found even by repeated punctures; or it may be obtained at one time and not at another, although introduced in the same intercostal space, the difference in result being due to the direction in which the needle is passed into the lung. When pus is found, the diagnosis of a localised empyema is generally regarded as established, and it is not until the chest is opened that the mistake is discovered. The operator then comes upon the lung, which may or may not be adherent. If the abscess follows an acute pneumonia the pus may show a pure culture of the pneumococcus. If it is due to a foreign body, there is invariably a mixed infection, and the pus is apt to be fetid.

When not treated surgically, abscess of the lung may rupture into the pleural cavity, producing a secondary empyema, or spontaneous

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<sup>1</sup> Archives of Pediatrics, January, 1904.



evacuation may take place through a bronchus and recovery follow. When the cause is a foreign body rapid recovery often follows its expulsion by coughing. If the diagnosis is made and proper surgical treatment is instituted, recovery occurs in probably the majority of cases.

The general plan of treatment should be the same as in empyema. In a small proportion of cases aspiration may suffice for a cure. However, incision is usually necessary. If the pleura is not adherent, adhesions should be excited by packing the thoracic wound with gauze, and after a few days a second operation may be done. The lung should be opened with a blunt instrument, following the line of the exploring needle, and a drainage-tube inserted as in empyema, the subsequent treatment being the same as for that disease.

#### GANGRENE OF THE LUNG.

Pulmonary gangrene is rare in children, although probably more common than in adults. It is most frequently associated with pneumonia. It is usually circumscribed, and seldom diagnosticated during life.

**Etiology.**—All my cases have been in children under three years old, the youngest an infant of four months. Gangrene occurs for the most part in children who are ill-conditioned, feeble, or cachectic, and often follows one of the infectious diseases, particularly measles. Of nine cases which have come under my personal observation, six complicated acute broncho-pneumonia and one lobar pneumonia. Pulmonary gangrene has been present in three per cent of my autopsies upon cases of pneumonia. The immediate cause of the necrotic process is interference with the circulation in a part of the lung, which is usually due to thrombosis or embolism of some of the branches of the pulmonary artery. To this there is added the entrance of putrefactive bacteria. In some cases pulmonary gangrene may begin as a septic thrombosis, this infection originating in some process in a distant part of the body.

**Lesions.**—The lower lobes are more frequently affected than the upper, and the surface of the lung rather than the central portions.

Two forms of gangrene may be seen: the diffuse form, which affects a whole lobe, or even a whole lung; and the circumscribed form, which occurs in a number of small scattered areas. The latter is the variety usually seen in children. In the diffuse form the lung is of a dirty green or brown colour, moist, and emits a gangrenous odour. In the circumscribed form, when occurring in pneumonia, the parts affected are of a gray or green colour, usually wedge-shaped, with the base at the surface of the lung. In the early stage they are not softened, and have no gangrenous odour; later, both these conditions may be present, and masses of necrotic lung tissue may be found in a cavity with ragged walls, partly filled with foetid pus. Careful dissection will reveal, in many

cases, the presence of thrombi in the vessels leading to the gangrenous parts.

**Symptoms.**—There are but two distinctive symptoms of pulmonary gangrene: the gangrenous odour of the breath, and the expectoration of masses of necrotic lung tissue. In the cases associated with acute pneumonia, which include the majority of those seen, death nearly always takes place before there is any separation of the sloughs, and even before very active decomposition in the necrotic areas has occurred. Both the peculiar symptoms are therefore wanting, and the diagnosis is made only at the autopsy. This has been true of nearly all the cases which have come under my own observation. But these patients, with one exception, were infants. In older children, particularly in cases secondary to the entrance of a foreign body, the characteristic symptoms are more frequently seen, and there may be a third symptom—hæmorrhage. This is present in about one-fourth of the cases (Rilliet and Barthez), and may be fatal. The general symptoms associated with gangrene are those of profound asthenia, resembling the typhoid condition.

From what has been said, it will be evident that the diagnosis is very difficult. If the characteristic odour of the breath is present, conditions in the mouth from which it might arise must be excluded. The physical signs differ in no respect from those of ordinary cases of pneumonia. The termination is almost always in death. This is due not only to the condition itself, but to the circumstances in which it is seen.

**Treatment.**—The general treatment should be supporting and stimulating, as in all severe cases of pneumonia. For the local process but little can be done, except the inhalation of antiseptics, of which creosote and turpentine are undoubtedly the best.

#### ACQUIRED ATELECTASIS—PULMONARY COLLAPSE.

These terms are applied to a state of the lung resembling the foetal condition, but occurring in a lung which has once been expanded. It may be due to compression or to obstruction.

**Collapse from Compression.**—The principal cause of this form is pleuritic effusion. It may also be produced by pneumothorax, enlargement of the heart, pericardial effusion, deformities of the chest from rickets or Pott's disease, and tumours of the mediastinum or the thoracic wall. In these conditions, on account of the external pressure, the air vesicles are not filled, although the bronchi are pervious. After collapse has existed for a considerable time, changes may take place in the lung which render expansion difficult or impossible. Unless, however, there are pleuritic adhesions, expansion often takes place readily after many weeks and even months. The symptoms and signs are those of the original disease.

Treatment is available chiefly in that form which follows pleuritic effusion, and will be considered in the chapter on Empyema.

**Collapse from Obstruction.**—This is due to two factors: blocking of either the large or small bronchial tubes, and feeble inspiratory force. The importance of collapse from obstruction in the acute diseases of the lung in infancy has, I think, been exaggerated. Whenever a large or small bronchus is completely obstructed by a foreign body, the portion of the lung to which the bronchus is distributed gradually becomes collapsed. If it is one of the primary bronchi which is occluded, a whole lung may be collapsed; if one of the lobar divisions, an entire lobe; if one of the smaller divisions, only a small area. The collapse does not take place immediately, but the contents of the air vesicles are gradually absorbed by the blood. The collapsed portion is slightly depressed below the surface of the lung. It is of a dark-red colour, very vascular, and to the naked eye resembles a pneumonic area, which it may subsequently become.

Many writers explain the development of broncho-pneumonia from bronchitis of the smaller tubes, through the intervention of pulmonary collapse, assuming that the obstruction of the small bronchi, from swelling of their walls and the accumulation of secretion, produces the same result as the plugging of a bronchus by a foreign body. In my own autopsies I have found little support for this theory. In acute bronchitis of the smaller tubes the lumen is narrowed, but seldom enough to prevent the entrance of air. The result is usually emphysema, not atelectasis. Such, at least, has been the condition I have most frequently found in autopsies in the earliest stage of broncho-pneumonia following bronchitis of the fine tubes. There are very often groups of collapsed air vesicles surrounding pneumonic areas, but these are neither an essential nor a very important part of the lesion. Collapse of a large part of the lung, or even of a lobe, I have never seen, either in pertussis or in acute bronchitis.

There is seen in delicate or rachitic infants a form of collapse which comes on very gradually. It is accompanied by bronchitis affecting the tubes in the dependent part of the lung. It may resemble the congenital form of atelectasis. Under the microscope there is almost invariably found, accompanying the collapse, lobular pneumonia and bronchitis of the tubes in the affected regions.

The symptoms of acquired atelectasis are much the same as in the persistent congenital form. The respiration is rapid, and there may be inspiratory dyspnoea with deep recession of the chest walls, especially if there is rickets. There is also cyanosis of variable intensity. The temperature is not elevated, but frequently is subnormal. The physical signs are very uncertain. There is usually feeble respiratory murmur over the affected areas, occasionally accompanied by moist râles. The essential

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point of difference between these cases and those of congenital atelectasis is that in the former the patients are often strong at birth, crying and breathing well, giving no signs of anything wrong in the lungs until the general nutrition has suffered from some other cause.

The following is a fairly typical case: A female infant thirteen months old had been under observation for several months before death. During this period she suffered a great part of the time from mild bronchitis. The chest was extremely rachitic. The respiration was always accelerated, and on inspiration the lateral recession of the chest was at times extreme. There was occasionally seen slight cyanosis, and during the last few weeks it was constant. Death occurred quite suddenly. At autopsy there was found very marked vesicular emphysema of both lungs in front. Nearly the whole of both lower lobes were in a condition of collapse, and of a uniform grayish-purple colour. The posterior portion of the upper lobes was similarly affected, but to a less degree. With moderate force all of the collapsed areas could be completely inflated. Bronchitis was present, but the pleura was normal.

The treatment of these cases is the same as that outlined in the chapter upon Congenital Atelectasis.

#### EMPHYSEMA.

Pulmonary emphysema consists primarily in overdistention of the air vesicles. It may result in their rupture and the escape of air into the interlobular connective tissue of the lung. In infancy and childhood emphysema is usually associated with acute processes.

**Etiology.**—Cases of emphysema are divided into two groups which are due to quite different causes. In one group it is compensatory, and consists in overdistention of the air vesicles in certain parts of the lungs because the full expansion of other parts is prevented either because they are consolidated, as in pneumonia or tuberculosis, bound down by adhesions from old pleurisy, or subjected to external pressure, as from chest deformities due to Pott's disease or rickets. In these conditions it is probable that the emphysema is produced during inspiration. It may also be produced by the artificial inflation of the lungs of the newly born.

In the second group of cases emphysema is produced by obstructive expiratory dyspnoea or cough. It is seen in all forms of laryngeal stenosis, in acute bronchitis and broncho-pneumonia, in asthma, pertussis, and occasionally it is produced by any condition which requires deep inspiration and holding the breath. In bronchitis the obstruction may be caused by a swelling of the mucous membrane or by an accumulation of secretion. In this group of cases air enters the lung, but as it can not readily escape, the air vesicles are distended, sometimes to such a degree that their resiliency is almost entirely lost.

**Lesions.**—The most common form in early life is acute vesicular emphysema, which occurs when the force distending the air cells is only moderate. In this form there is dilatation of the vesicles with very slight structural changes, there being usually rupture of a few alveolar septa only (Fig. 73). Although the dilatation may be quite marked, the emphysema is not permanent. The parts most affected are the upper lobes, particularly the anterior borders. In appearance the emphysematous lung is pale, sometimes almost white. The areas are prominent, and do not collapse upon opening the chest. With a lens, or even with the naked eye, the individual air vesicles can often be distinguished as minute pearly bodies, at times resembling miliary tubercles. When the disease is secondary to acute bronchitis or laryngeal stenosis it may affect nearly the whole of both lungs.

With a greater distending force rupture of many of the air vesicles results, and this may give rise to interstitial or interlobular emphysema. At times blebs are formed, varying in size from a pin's head to a cherry or even larger. These are usually seen at the anterior border or at the root of the lung on its inner surface. Again, the air finds its way between the lobules, dissecting them apart in all directions throughout the lung. Sometimes a large part of the surface of both lungs is seamed with irregular deep crevasses containing air, the largest being an inch or more in length and nearly one-fourth of an inch wide. The most severe cases occur in pertussis. On two or three occasions I have seen this form of emphysema, once to an extreme degree, where children had died from diseases unconnected with the respiratory tract, and where no history could be obtained which threw any light upon the etiology of the emphysema. Rupture of the blebs which form at the root of the lung may lead to emphysema of the mediastinum, or even of the subcutaneous connective tissue of the body. This is occasionally seen in whooping-cough and in laryngeal stenosis. The primary or substantive form of emphysema seen in adult life rarely if ever occurs in childhood.

**Symptoms.**—Emphysema occurring in acute pulmonary diseases gives rise to no peculiar symptoms and to no physical signs except exaggerated resonance upon percussion. This masks dulness from consolidation and also that from the liver and spleen. If the patients recover from the original disease, the emphysema greatly diminishes or disappears completely in the course of a few weeks or months. Acute interlobular emphysema can not be diagnosticated during life, unless, as is sometimes the case, general subcutaneous emphysema is seen, which may come on quickly, last for several hours or days and then gradually disappear.

The treatment of emphysema is that of the disease with which it is associated.

CHAPTER VI.

*PLEURISY.*

ALL the common forms of inflammation of the pleura are seen in childhood. In the great majority of cases they are secondary to disease of the lung itself. Serous effusions are much less frequent than in adults, and under three years they are rare. Purulent effusion (empyema) is, however, much more often seen than in adult life, and it is the most important variety of pleurisy with which the physician has to deal.

Whether inflammation of the pleura ever occurs as a strictly primary disease is still a mooted point. Cases are occasionally observed clinically in which both the serous and purulent forms of the disease appear to be primary, but these are extremely rare. Acute pleurisy may, however, follow inflammation of the lung so rapidly that it is not easy to determine that the lung was first affected. In infants, extension from the lung is almost the sole cause. It occurs both with lobar and bronchopneumonia, existing to some degree in nearly every case in which there is consolidation of the lung. Next in frequency to simple pneumonia as a cause of pleurisy are the tuberculous processes of the lung. Tuberculous pleurisy without tuberculosis of the lungs or the bronchial glands is of doubtful occurrence. Acute pleurisy is not an infrequent complication of the infectious diseases, particularly scarlet and typhoid fevers, measles, and influenza. In most of these cases also it is secondary to disease of the lung. Pleurisy in older children occasionally follows cold and exposure, although it is doubtful whether in any case this is the only cause. In them also it may occur as a complication of rheumatism.

The most important cause of acute pleurisy being extension from pneumonia, it follows that it is most frequent in the cold season, that it occurs more often in males than in females, and between the ages of one and five years. It may, however, be seen at all ages, and may even occur in intra-uterine life. The youngest case in which I have found extensive pleuritic adhesions as an evidence of previous inflammation was in an infant of three months. In this case firm connective tissue adhesions were found over the whole of both lungs.

DRY PLEURISY.

In infants and young children this usually accompanies pneumonia or tuberculous processes in the lung. In older children it may be primary.

**Lesions.**—On account of the frequency with which this occurs in pneumonia we have an opportunity of observing it in all stages. In the mildest varieties it affects only the pulmonary pleura, and occurs over the

pneumonic areas. The pleura is injected, has lost its lustre, and appears dull or roughened. This is due to an exudation of fibrin upon its surface. If the process continues, more fibrin is poured out, and there are in addition swelling and a proliferation of the connective-tissue cells, and an exudation of leucocytes from the blood-vessels. The pleura is then coated with a layer of fibrin of variable thickness, in which are entangled pus cells and new connective-tissue cells. The layer of fibrin varies from the thickness of tissue paper to that of an ordinary book cover. In recent cases it may easily be stripped off, while in older ones it becomes organised and is firmly adherent. The colour of the exudate varies with the number of pus cells. It is gray, grayish-yellow, or yellowish-green, according as these cells are few or numerous. As a rule, dry pleurisy is localised, but the two opposing surfaces are affected. Part of the exudate is usually absorbed, but it is doubtful if complete recovery occurs, there being left behind some adhesions between the visceral and parietal layers.

In the dry form of tuberculous pleurisy there may be only an exudation of fibrin, or the pleura may be covered with gray tubercles and yellow tuberculous nodules. These are not only seen upon the pleura, but develop in the exudation. In this form, which is usually chronic, great thickening of the pleura may take place. Both the serous and purulent effusions occurring in conjunction with tuberculosis are likely to be sacculated because of the previous existence of adhesions.

After nearly every case of dry pleurisy there probably remains some slight thickening of the pleura. In certain cases there follows a chronic inflammation of the pleura with the production of new connective tissue, which results in thickening and adhesions, which may be so extensive as to entirely obliterate the pleural cavity. Either one or both sides may be affected. It is usually accompanied by external pericarditis. This form is extremely rare in childhood.

**Symptoms.**—As an independent clinical disease, acute dry pleurisy has no existence in infancy or early childhood. The cases which are occasionally so diagnosticated have in my experience invariably proven to be broncho-pneumonia. In children from ten to fourteen years old, dry pleurisy may occur under the same conditions as in adults.

The symptoms are sharp, localised pain, increased by full inspiration, sometimes tenderness upon pressure, and a short, teasing cough. The pain is not always felt upon the affected side, and it may be referred to the abdomen. Upon physical examination, dry pleurisy is recognised by the presence of a pleuritic friction sound. This is usually of a moist, crackling character, generally localised, and heard both on inspiration and expiration. It is quite superficial, and not changed by coughing. This form of pleurisy, as a rule, runs a course of a few days or a week, without constitutional symptoms. When dry pleurisy occurs as a com-

plication of pneumonia it is recognised by the signs just mentioned; but it usually causes no new symptoms except pain.

**Treatment.**—The treatment consists in counter-irritation by mustard, iodine, or blisters, according to the severity of the inflammation, and in the use of opium. Severe pain can sometimes be relieved by firmly encircling the chest with a broad band of adhesive plaster.

#### PLEURISY WITH SEROUS EFFUSION.

This form of pleurisy is not common in young children, and in infants it is rare. It usually occurs as a complication of pneumonia, but may be seen in nephritis, acute rheumatism, scarlet fever, or any of the other acute infectious diseases. It may be tuberculous. In rare cases it appears to be primary. Bacteria are occasionally present in the exudation, even in cases which do not become purulent, but their number is usually small. The pneumococcus, the streptococcus, and the tubercle bacillus are the forms most often seen.

**Lesions.**—The early changes are much the same as in dry pleurisy, but in addition serum is poured out from the blood-vessels, in some cases almost from the beginning of the inflammation. This may be small in amount, or it may fill the pleural cavity. The lesions are similar to those seen in adults, except that in children there is apt to be more fibrin. The process usually terminates in absorption of the serum, but, as in dry pleurisy, more or less extensive adhesions are left behind from the fibrinous exudation. In other cases there is at first a clear serum, often containing pneumococci, then it becomes somewhat turbid, and finally purulent. This is especially common in infants.

**Symptoms.**—The small serous effusions of one or two ounces, occurring with the pleurisy that complicates pneumonia, rarely cause either symptoms or physical signs by which they can be recognised. In the present connection only those cases will be discussed in which the amount of effusion is considerable. This form of pleurisy sometimes follows a well-defined attack of pneumonia. Other cases come on with acute febrile symptoms somewhat resembling those of pneumonia, but with all the symptoms less severe, except the pain. After an illness of only two or three days the chest may be found full of fluid. In a third class the disease comes on insidiously, with little or no fever, and often with no distinct pulmonary symptoms except shortness of breath. There are general weakness, sometimes loss of flesh, anæmia, and moderate prostration; but usually the patients are not sick enough to go to bed. The symptoms of pleurisy with effusion vary greatly. When it occurs as a complication of some acute infectious disease, it is often latent, and the diagnosis is to be made only by the physical examination of the chest.

In cases in which the fluid does not become purulent, the usual course



of the disease is for the fluid to disappear gradually by absorption, the case going on to spontaneous recovery. Serious symptoms resulting from pressure upon the heart and lungs are not common, but may occur when the fluid accumulates rapidly; hence they are most likely to be seen early in the attack. There may be great dyspnoea, sometimes orthopnoea, cyanosis, weak pulse, and even attacks of syncope. Death may occur with these symptoms. In certain cases there is seen no tendency to spontaneous absorption, and the exudation may remain stationary for months. There may then be fever, usually slight but sometimes quite regular, with a decline in the general health, pallor and anæmia, which may strongly suggest the existence of pus, although this is not present. Others are regarded as cases of tuberculosis.

**Physical Signs.**—The signs in the chest are essentially the same whether the fluid is serous or purulent. On inspection, there is diminished movement of the affected side, sometimes bulging of the intercostal spaces, and if the effusion is large, an increase in the measurement of the affected side of the chest. The apex beat of the heart will usually be considerably displaced if the effusion is upon the left side. It may be found at the epigastrium, at the right border of the sternum, or even in the right mammary line. In disease of the right side the displacement is less, and occurs only with a large effusion. It may then be found in or near the left axillary line. On palpation, the vocal fremitus is usually diminished or absent, but it may be but little changed. Percussion gives marked dulness or flatness. In a large effusion this is over the entire lung. There is also a sensation of increased resistance appreciable by the percussing finger. With a smaller effusion there is usually flatness over the lower part of the chest and dulness or tympanitic resonance above; sometimes dulness is found behind and tympanitic resonance at the apex in front. The line of flatness may change with the position of the patient. Grocco's sign is found in the majority of cases. This is a triangular area of dulness posteriorly, with its base to the spine, on the side opposite to the effusion. The signs on auscultation are variable, and probably lead to more frequent mistakes in diagnosis than in any other pulmonary affection. Bronchial breathing and bronchial voice over the fluid are common in children. Absence of both voice and breathing is sometimes met with, but it is exceptional. The bronchial breathing over fluid usually differs from that over consolidation, in that it is feebler and distant; in some cases, however, it is indistinguishable from that heard over consolidation. Friction sounds may be heard above the level of the fluid, or when the fluid is subsiding, and there may be bronchial râles.

**Diagnosis.**—The most reliable signs for diagnosis are displacement of the heart, flatness on percussion, absence of râles and friction sounds, and (usually distant) bronchial breathing. In an infant, flatness should always lead one to suspect fluid. If there is flatness over one entire

lung, the existence of fluid is almost certain. Between serous and purulent effusions a positive diagnosis is possible only by the use of the exploring needle. This should be employed in every case, as for treatment it is important to know at once whether or not we have a purulent effusion to deal with. The amount of fluid in serous pleurisy is generally less than in the purulent variety.

Pleurisy is further to be differentiated from pneumonia, and from tuberculosis. From pneumonia, the acute cases are distinguished by the lower temperature, the less severe prostration, and the fact that all the general symptoms are milder, but especially by the physical signs. The differential diagnosis by the physical signs between effusion and the various forms of consolidation is considered under the head of Empyema.

**Prognosis.**—In the acute cases complicating pneumonia, a serous pleurisy is very apt to become purulent. Other forms of pleurisy with effusion, as a rule, terminate in recovery. In cases coming on without definite cause there should always exist a suspicion of tuberculosis, and hence every patient should be closely watched for the development of the other signs of that disease.

**Treatment.**—In the great majority of cases, only symptomatic treatment is required during the acute period. The patient should be kept in bed, and pain relieved by opium, counter-irritation, or dry cups. After the fever has ceased the patient may be allowed to sit up, but all exertion should be carefully avoided if the effusion is large. Sudden death has occurred when this rule has been violated. The patient should in suitable weather be kept in the open air as much as possible. In the course of a few weeks the effusion usually subsides under simple tonic treatment. Absorption may sometimes be hastened by counter-irritation and diuretics; but convalescence is apt to be slow, and it may be several months before the health is entirely restored.

The removal of the fluid by operation is indicated in the acute form when it is accumulating so rapidly as to endanger life from the pressure upon the heart and lungs; also when there is no tendency to absorption after from two to three weeks of constitutional treatment. In such cases nothing is to be gained by waiting, and harm may be done to the lung by the delay. The usual method is by aspiration. In the acute stage enough should be removed to relieve the patient's symptoms, aspiration being repeated if necessary in twelve or twenty-four hours. In infants, particularly, there is great danger of wounding the lung when aspiration is repeated several times. This usually results in the production of pneumo-thorax which may mask the re-accumulation of the fluid. In the subacute stage the removal of a portion of the fluid may be all that is required, spontaneous absorption of the remainder often taking place quite promptly. A few cases of serous pleurisy have been incised and drained as cases of empyema.

## EMPYEMA.

Fully nine-tenths of the cases of empyema in children under five years either occur with or follow pneumonia, being usually the sequel of the form described as pleuro-pneumonia. In some of these cases, however, the pleurisy masks the pneumonia, so that the former appears to be the primary disease. Tuberculosis is a rare cause in early childhood, but becomes more frequent after the seventh year. Empyema may complicate scarlet fever, measles, or any of the other acute infectious diseases. It is met with in pyæmia from all causes. It may occur in the newly born as the result of infection through the umbilical wound or the skin. It is seen with suppurative inflammations of the joints and in osteo-myelitis. It may complicate suppurative processes in the abdomen, such as appendicitis or purulent peritonitis. Among the local causes may be mentioned traumatism, necrosis of a rib, and the rupture into the pleural cavity of abscesses originating in the mediastinum, in the thoracic wall, or below the diaphragm.

**Etiology.**—Since empyema is generally secondary to pneumonia, its causes are mainly those of that disease. Bacteriologically, the cases may be divided into several groups:

1. Those containing the pneumococcus (*micrococcus lanceolatus*), usually in pure culture. This is the largest group, and includes nearly all the cases secondary to pneumonia.

2. Those containing other pyogenic germs, particularly the streptococcus and the staphylococcus aureus. These organisms may be found alone, or associated with the pneumococcus. This combination is likely to be found in cases secondary to the pneumonia which occurs with the infectious diseases. The streptococcus and staphylococcus occur in the pleurisy of pyæmia, and generally also when the disease is due to the rupture of abscesses into the pleural cavity. I have once found the influenza bacillus as the sole organism in empyema.

3. The cases due to tuberculosis. These are rare in children and almost unknown in infants. The tubercle bacillus is often difficult to demonstrate, and it may be absent. But it is not safe to assume that tuberculosis is present because no organisms are found.

**Lesions.**—Empyema is an inflammation with the production of serum, fibrin, and pus. In most of the cases—and the younger the child the more frequent its occurrence—it succeeds pleuro-pneumonia. There is first an exudation of fibrin with an excess of pus cells. As the process continues, more and more pus is poured out, with serum. At first the fluid collects in small pockets formed by the slight adhesions. As it accumulates these are broken down, and the pleural cavity may be filled with pus. If the original inflammation involved but a portion of the pleura the empyema may be sacculated. This is often seen even in

infants. Sacculated empyema is usually posterior and over one lower lobe, but may be in any part of the chest. In very rare cases there may be several sacs containing pus, separated by septa. This I have never seen in empyema following pneumonia. The cases just described are those in which, in infants and young children, the pneumococcus is regularly found. The amount of fibrin is large, covers both surfaces of the pleura, and many large masses float in the fluid. The pus is usually thick, creamy, and odourless. In another group of cases the evidences of inflammation of the pleura are much less marked, and in some they may be slight. There is but little fibrin in the exudate, and adhesions are rare. In this form the streptococcus or the staphylococcus are the organisms usually found. In these cases the inflammation may be purulent from the outset, and the pus is thinner than in the preceding variety. Empyema following pneumonia is occasionally preceded by a serous effusion which, although almost clear, is usually found to contain great numbers of bacteria, usually pneumococci.

Even when the fluid is moderate in quantity it is not all at the bottom of the chest, but is generally distributed over a considerable part of its surface, and its depth at the middle and upper part of the chest may be only half an inch, or even less. When the accumulation is larger, the lung does not float on the surface of the fluid, but the fluid surrounds the lung, which is compressed on all sides (Fig. 93). The heart is displaced; the diaphragm and the abdominal viscera are somewhat depressed, and there may be bulging of the chest on the affected side. The amount

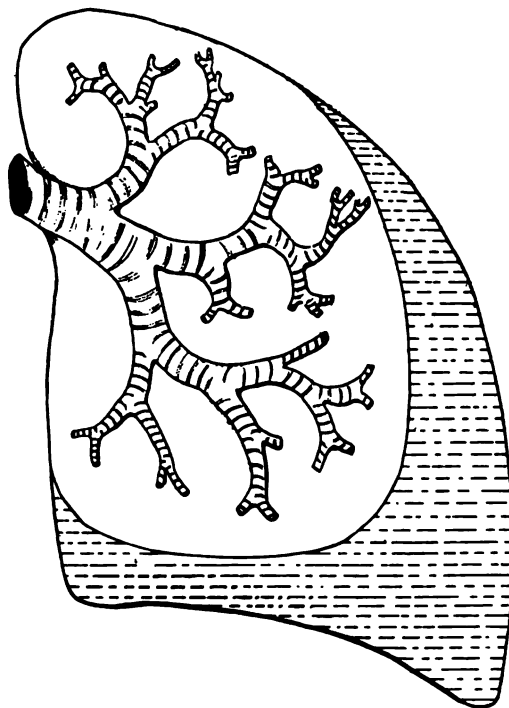


FIG. 93.—SECTION OF A LUNG. To illustrate the distribution of the fluid in the chest in a moderately large effusion (diagrammatic).

of fluid in ordinary cases is from four to twenty ounces, although in neglected cases it may accumulate until it amounts to four or five pints. The effect upon the lung will depend upon the amount of fluid and the duration of the compression. When the quantity is small, or when the

pressure is removed early, the lung in most cases readily expands, air being forced into it from the opposite lung, especially during the act of coughing. With the exception of adhesions, recovery may be complete. Although wide in extent, the adhesions are not usually strong enough to interfere seriously with the function of the lung. If the pressure is great and has been long continued, the adhesions over the lung may become so dense and firm that expansion is difficult, and can at best be only partial. In such cases recession of the chest wall occurs. In old cases, expansion is still further interfered with by the changes taking place in the lung itself, usually a low grade of interstitial pneumonia.

In cases receiving no treatment, absorption of the pus is possible, but is not to be expected. It generally seeks an external outlet; the lung may be perforated and the pus evacuated through the bronchi, or external rupture may occur, generally in the neighbourhood of the nipple. In still other cases the pus may burrow along the spine, or through the diaphragm reaching the peritonæum.

Empyema is more often of the left than of the right side, the proportion being about three to two. It is double in about three per cent of all cases, but much oftener in infants. The most serious complication in young children is pericarditis, usually with empyema of the left side; in older children the most frequent complication is pulmonary tuberculosis.

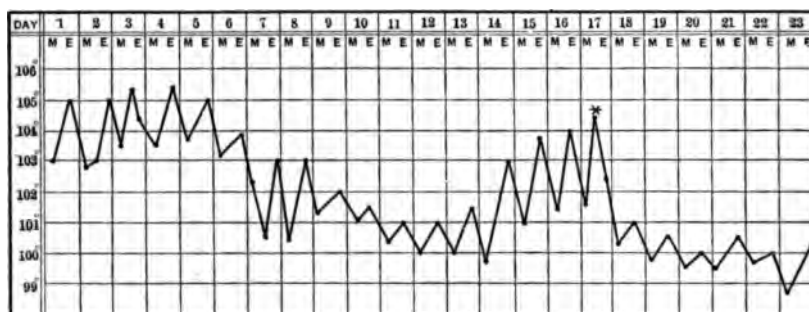


FIG. 94.—EMPYEMA FOLLOWING PNEUMONIA. Private patient, girl, eight years old; severe pneumonia terminating by lysis; development of empyema indicated by secondary temperature; operation on seventeenth day; recovery.

**Symptoms.**—When it occurs as a sequel of pneumonia, the symptoms of empyema may follow those of the original disease without any intermission; or after the temperature has been normal or nearly so for several days it may rise again, sometimes quite suddenly, but more often gradually. With this accession of fever there are other symptoms pointing to an increase in the thoracic disease. (See Figs. 94 and 95.) After scarlet fever or other infectious diseases, the onset of empyema is often signalled by cough, rapid breathing, and the other usual symptoms of pulmonary disease. In the cases where empyema appears to be

primary, the onset is sudden, with high temperature and general and local symptoms resembling those of pneumonia. After such a beginning, the chest may be found full of pus by the third or fourth day. In older children empyema may come on with gradual, and even insidious symptoms, there being only slight fever, dyspnoea, and cachexia. Marked leucocytosis, 30,000 to 50,000, is almost invariably present. The proportion of polymorphonuclear cells is usually from seventy to eighty per cent.

Whatever may have been the mode of onset, when the pus has been in the chest for some time the symptoms are fairly uniform. During the

acute stage there are present pallor, anæmia, and prostration. The respirations are always accelerated, being usually from forty to seventy a minute. Cough is present; there is dyspnoea, sometimes marked, but more often it is scarcely noticeable. The temperature is exceedingly variable; usually it ranges from 101° to 103° F. A typical hectic temperature with sweating, is in my experience very rare. The pulse is rapid but of fair strength. There is loss of flesh, sometimes even emaciation and anorexia; occasionally there is diarrhoea. The stage of acute symptoms may last from two to four weeks. This may be succeeded by a subacute stage which may last for months. In this there is little or no fever; the patient seems convalescent so far as regaining strength and colour are concerned; but cough, dyspnoea, and rapid respiration continue. The chest shows no change in signs from those of the acute stage. In chronic cases the general symptoms closely resemble those of tuberculosis. There may be clubbing of the fingers, albuminuria, swelling of the feet, and often marked lateral curvature of the spine.

**Diagnosis.**—The physical signs do not differ essentially from those present in serous effusion. If there are signs of fluid in the chest and the patient is under three years of age, the fluid is usually purulent; and from the third to the seventh year, pus is much more often found than serum. A marked leucocytosis always makes pus more probable. In every case in which fluid is suspected the exploring needle should be used, because of the great importance of an early diagnosis. The skin should be surgically clean and the needle sterilised. Pus may not be

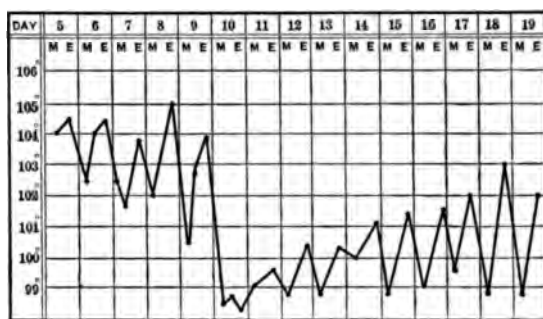


FIG. 95.—EMPYEMA FOLLOWING PNEUMONIA. Hospital patient, two years old; single-lobe pneumonia with crisis on ninth day; no resolution, but instead gradual development of signs of empyema closely following the temperature curve.

found because the needle is too small, too short, or because it is introduced too far into the chest; for when the layer of pus is thin, the needle may be pushed through this into the lung.

The physical signs upon which most reliance is to be placed are, marked dulness or flatness on percussion, feeble breathing, and displacement of the heart. When in a young child these signs are present, whether general or localised, a needle should be inserted, and if pus is not found at the first trial, repeated punctures should be made until the presence or absence of fluid is definitely settled.

Empyema is most frequently confounded with unresolved pneumonia. The differential points are that in unresolved pneumonia the dulness is usually over a single lobe, râles or friction sounds are heard, and there is no displacement of the heart; empyema may give flatness over the whole lung, or over the lower half of the chest in front and behind, râles and friction sounds are absent over this area, and the heart is usually displaced. In both conditions we may get bronchial breathing and voice. The confusion of acute pneumonia or tuberculosis with empyema, generally arises from placing too much reliance upon auscultation. In pleuro-pneumonia, with an excessive exudation of fibrin, the signs may be identical with those of empyema, except that the heart is not displaced. I have twice seen pulmonary tuberculosis, with caseation of an entire lobe, which gave signs that were identical with those of a sacculated empyema. It is by the exploring needle, and by that alone, that empyema is positively differentiated from these pulmonary conditions.

There are some other thoracic diseases from which the diagnosis may be even more difficult. A large pericardial effusion gives signs which are in some cases identical with those of empyema of the left side. Marked displacement of the heart to the right is always a strong point in favour of empyema; besides, such pericardial effusions are extremely rare in young children. A pulmonary abscess of considerable size—also a rare condition—gives signs identical with those of localised empyema, and is only distinguished from it by autopsy or operation. Abscesses from broken-down tuberculous glands may give signs resembling those of localised empyema, and like an empyema may point between the ribs in the upper part of the chest. The constitutional symptoms of empyema may at times resemble typhoid fever or malaria; but it is distinguished from them by the physical signs and by the examination of the blood.

**Prognosis.**—The outcome of a case of empyema depends chiefly upon the age and general condition of the patient, the exciting cause, the duration of the symptoms, the presence or absence of serious complications, and the treatment. The mortality in infants under one year, particularly hospital cases, is very high—fully ninety per cent. It is difficult to understand why these cases do so badly; many of these children

on admission are in excellent condition and do well for a week or more after operation. Then the temperature rises, the patients lose ground rapidly and die of exhaustion at the end of the second or third week. Their inability to expand properly the compressed lung has always seemed to me an important factor, as this condition is almost invariably found at autopsy. Very seldom is there trouble with drainage. Empyema in children over two years old seen reasonably early and receiving proper treatment, almost invariably terminates in recovery, unless the disease is double or serious complications exist. The best results are seen in the cases that follow pneumonia. Pneumococcus and staphylococcus cases have a better outlook than those due to the streptococcus. Tuberculosis before the seventh year is an exceedingly infrequent cause, and gangrene of the lung and general pyæmia are both rare causes in early life. It is these three conditions that make the prognosis of the disease in adults so serious. Great delay in operation makes the prognosis worse, because the more difficult the expansion of the lung the more tedious is the disease, and the greater the likelihood of a sinus remaining. With proper early treatment these patients not only recover, but in most cases the recovery is surprisingly complete. Retraction of the chest and its resulting lateral curvature of the spine are rare, and seen only in neglected cases. In very many cases, in which a reasonably early operation was done, it is impossible, after the lapse of two or three years, to detect any difference whatever in the physical signs of the two sides of the chest. There are few serious diseases the treatment of which is more satisfactory than that of acute empyema in older children.

Spontaneous recovery in empyema may take place by absorption; but this is so rare that it is not to be expected. The pus may be evacuated spontaneously through a bronchus, rupture having taken place through the visceral pleura. When this occurs, a large amount of pus may be coughed up in a few hours, usually followed by immediate, but not always lasting, improvement. This is the most favourable of the natural terminations. External opening may take place, usually in the region of the nipple. There is an area of redness, then a fluctuating tumour, and finally the pointing of an abscess. The discharge may continue for months, or even for years. External opening rarely occurs until the disease has lasted several months. Of 19 cases of empyema in children collected by Schmidt, in which a spontaneous discharge of pus occurred either externally or through a bronchus, there were 17 deaths and 2 recoveries. Empyema may burrow behind the diaphragm into the abdominal cavity, appearing as a psoas abscess; it may burrow posteriorly into the lumbar region; it may rupture into the œsophagus, or through the diaphragm into the peritoneal cavity. All these conditions, however, are very rare. The chances of spontaneous cure in empyema are small. Of 32 cases, reported by Rilliet and Barthez, which received



no surgical treatment, 21 proved fatal. The statistics of empyema before the general adoption of surgical treatment are appalling. Patients were either worn out by the protracted suppuration, or died from amyloid degeneration, pneumonia, or tuberculosis.

**Treatment.**—The medical treatment relates to the patient only; the disease is always to be treated surgically. Like any other acute abscess, empyema requires free incision and drainage with proper aseptic precautions.

*Aspiration* as a means of cure is now seldom used. Unquestionably it sometimes suffices to cure empyema, most frequently when it is localised, and when the cause is the staphylococcus. How often this occurs is shown by the following statistics: Of 139 cases which I collected that were treated by aspiration, 25 were cured, 8 of these by a single aspiration; 13 died, and the remaining 101 were afterward subjected to other treatment. The objections to aspiration are, that it is not possible to remove all the pus; that it affords no opportunity for the removal of the large fibrinous masses; besides, there is the danger, especially with repeated aspirations, of puncturing the lung and producing pneumothorax. Simple aspiration, therefore, is to be advised in children only for temporary relief when the amount of fluid is large and the symptoms are urgent. Aspiration, followed by the injection of formalin and glycerine, is not, from my experience, to be recommended. Likewise, the procedure of continuous aspiration, as proposed by Bryant, I have found in infants equally unsatisfactory.

*Incision and Drainage.*—In many cases it is preferable to delay incision until the period of most acute inflammation has subsided, as shown by lower temperature and stationary physical signs. This is usually seen two or three weeks after the pleural invasion. Such delay is not admissible if either the local condition or the temperature points to a steady increase in the disease; nor when the general symptoms indicate increasing prostration or sepsis. The dangers attendant upon general anæsthesia are considerable, and in most cases it is better not to employ it. I have known of four deaths on the table during operation, and in several other cases have seen very alarming symptoms occur. Chloroform is more to be feared than ether. It is well, when possible, to employ local anæsthesia. The most favourable point for incision is the posterior axillary line in the seventh intercostal space upon the right side, the eighth upon the left. In a case of localised empyema, the lowest point at which pus can be obtained by puncture should be chosen. The incision is made in the middle of the intercostal space. No matter what has been found by puncture on previous occasions, the exploring needle should always be used at the time of operation and at the site of the incision before the latter is made. The incision should be only large enough to allow the introduction of two tubes

side by side into the pleural cavity. The hæmorrhage is very rarely sufficient to require a ligature. It is, I believe, undesirable to attempt to empty the chest at the time of operation. A better plan is to insert the tubes at once and apply the dressings, allowing the pus to escape slowly. The drainage tubes should be of rubber, fenestrated, one-fourth to three-eighths of an inch in diameter and about three inches long. To secure them from slipping into the cavity, the outer end should be transfixed by a large safety-pin before introduction.

Both the original operation and the subsequent dressings should be done with strict aseptic precautions on account of the danger of secondary infection, the occurrence of which adds to the severity and prolongs the course of the disease. After the third or fourth day the second tube may be omitted and the remaining one gradually shortened. Often, by the end of the fourth week, and sometimes before, the tube may be dispensed with altogether. The time of redressing and the removal of the tube is determined by the amount of discharge and the temperature.

Simple incision with drainage I believe to be the preferable operation for recent cases in infants. One advantage over rib resection is that it is shorter and altogether less of an operation, these factors being at times of considerable importance in very young and feeble children. Again, it can be done without an anæsthetic, and it has seemed to me that pulmonary expansion took place with greater facility than when the much larger opening into the chest was made. Finally, in a large number of cases, it gives all the room needed for drainage. There are, however, some disadvantages. The smaller opening may not give adequate room for the removal of large masses of fibrin. In old cases, particularly, it not infrequently happens that after the chest has been emptied the ribs become so closely approximated that but little space is left, and the drainage tubes are pinched. Furthermore, the contact of the tubes may lead to erosion and superficial necrosis of the adjacent ribs, sometimes to exostoses. While, therefore, simple incision with drainage is to be preferred in the case of infants, for all other patients the resection of a rib seems desirable and advantageous. The removal of an inch of rib is usually all that is necessary.

Kenyon's method of continuous drainage into a wash-bottle below the water level, has much to commend it especially for infants. The opening made into the chest is a small one admitting only a single tube. The wound is tightly packed about the tube so as to admit no air. The thoracic tube is connected by several feet of rubber tubing with the wash-bottle which contains a sterile salt solution. This bottle is suspended beneath the patient's bed or placed upon the floor. The character and the amount of discharge can thus readily be seen. As the tube need not be changed for several days the child is spared the fatigue and distress of frequent dressings. The small opening into the chest is

of considerable advantage in preventing the admission of air; it thus diminishes the danger of secondary infection and favours the expansion of the lung. Should the tube become blocked it can be cleared by raising the bottle and allowing the fluid to flow into the chest and then siphoning it out. The bottle is emptied once or twice a day and the air is excluded by clamping the tube meanwhile.

Washing out the pleural cavity is indicated in cases in which the pus is foul. A single washing for the purpose of removing fibrin is

the routine practice of some surgeons. For this a warm, sterilised salt solution should be used. Personally, I have seldom found this necessary. Repeated irrigations should not, I think, be employed. The usual duration of the discharge in cases treated by simple incision is from three to six weeks, the average being about five weeks.

A persistence of temperature or a fresh rise after operation most frequently indicates defective drainage, generally due to blocking of the tube; but this is not always the case. It may be due to pneumonia, either a continuance of the old process or the lighting up of a new one, to abscess of the lung, to empyema of the opposite side, to pericarditis, or to some cause outside the chest, very frequently otitis. The mistake is often made of allowing the tube to remain for too long a time, so that a sinus is kept open which would otherwise close.

In chronic cases, or those which have been long neglected, some further operative treatment is often necessary. The lung is so bound down by firm adhesions that further expansion is impos-



FIG. 96.—DEFORMITY AFTER AN OLD EMPYEMA OF THE LEFT SIDE FOR WHICH ESTLANDER'S OPERATION WAS PERFORMED. Portions of five ribs were removed. (From a photograph seven years after operation.)

sible, and even after the chest has receded to its utmost, so that the ribs are in contact, there still remains a cavity which can not close. For such

cases the only hope is an operation by which portions of several ribs are removed, thus allowing a greater collapse of the chest wall. This is known as *thoracoplasty*, or *Estlander's operation*. The operation is of itself a serious one, and only to be advised as a last resort in inveterate cases. Such an operation is, of course, always followed by very great deformity (Fig. 96).

*Methods of Inducing Expansion of the Lung.*—

In most of the cases, particularly the recent ones, complete expansion of the lung takes place without any difficulty, the chief agent being the cough. In some cases this may be insufficient. The apparatus, devised by James, shown in the accompanying cut (Fig. 97), serves at the same



FIG. 97.—JAMES'S APPARATUS FOR EXPANDING THE LUNG AFTER EMPYEMA.

time as a toy for the child's amusement and as a most efficient means of inducing forced expiration. One bottle is placed a few inches higher than the other, and the child blows a coloured fluid from the lower into the higher bottle, allowing it to siphon back. Blowing soap bubbles often answers the same purpose.

SECTION V.  
DISEASES OF THE CIRCULATORY SYSTEM.

CHAPTER I.

*PECULIARITIES OF THE HEART AND CIRCULATION IN EARLY LIFE.*

**The Fœtal Circulation.**—During the latter part of fœtal life the circulation may be briefly described as follows: The purified blood comes from the placenta through the umbilical vein. Entering the body, it divides at the under surface of the liver into two branches, the smaller one, the ductus venosus, communicating directly with the inferior vena cava; the larger branch joining the portal vein, so that its blood traverses the liver, and then enters the inferior vena cava through the hepatic vein. From the inferior vena cava the blood enters the right auricle, like that returned from the head and upper extremities by the superior vena cava. A part of the blood now passes directly into the left auricle through the foramen ovale; the remainder, through the tricuspid orifice into the right ventricle. As the requirements of the pulmonary circulation are not great, only a small part of the blood is sent through the pulmonary artery to the lungs; the greater portion passes from the pulmonary artery through the ductus arteriosus into the aorta, joining here the blood from the left ventricle. The blood thus finds its way from the right heart to the left, only in small part by way of the lungs, the greater part passing directly from the right auricle to the left, or from the right ventricle into the aorta through the ductus arteriosus. From the aorta, the blood reaches the placenta through the umbilical arteries, which are a continuation of the hypogastric arteries, which in turn are given off from the internal iliacs.

**Changes in the Circulation at Birth.**—With the ligation of the umbilical cord, the circulation through the umbilical vein and arteries and the ductus venosus ceases. With the establishment of respiration and the consequent increased demands made by the pulmonary circulation, the blood ceases almost at once to pass through the ductus arteriosus, and very soon through the foramen ovale. The umbilical vessels during the first few days of life are filled with small thrombi, which become organised. By the end of the first week, these vessels, as well as the ductus venosus, are usually closed at their extremities, although they may remain patulous throughout the greater part of their extent for several weeks. They subsequently atrophy to the condition of small fibrous

cords. For some weeks before birth the circulation through the foramen ovale is slight, it being gradually obstructed by the growth of a septum which nearly fills the space at birth. After the first week of extra-uterine life very little, if any, blood passes through it, although complete closure of the foramen often does not take place until the middle of the first year. In fully one-fourth of the autopsies I have made upon infants under six months old, there have been found minute openings at the margin of the foramen ovale, but they are usually oblique, and closed by the valvular curtain so as effectually to obstruct the current of blood. The ductus arteriosus is first closed by a clot, which becomes organised and blends with the products of a proliferating arteritis. It is rarely found open after the tenth day, and by the twentieth it is almost invariably obliterated.

**The Pulse.**—The pulse in early life is not only more frequent, but it is very much more variable than in adults. The following is the average pulse-rate in healthy children during sleep or perfect quiet:

|                                |                        |
|--------------------------------|------------------------|
| Six to twelve months .....     | 105 to 115 per minute. |
| Two to six years .....         | 90 " 105 " "           |
| Seven to ten years .....       | 80 " 90 " "            |
| Eleven to fourteen years ..... | 75 " 85 " "            |

The pulse is a little more frequent in females than in males, and more frequent when sitting than when lying down. Muscular exercise or excitement increases the pulse-rate by from twenty to fifty beats. Very trivial causes disturb not only the frequency but the force of the pulse. The pulse in young infants may be irregular even in health and during sleep. When rapid, it is frequently irregular without special significance. No diastole is seen in the pulse wave of early infancy.

The circulation is much more active in infancy than in later childhood; thus, according to Vierordt, the entire round of the circulation is accomplished in the newly born in twelve seconds; at three years, in fifteen seconds; in the adult, in twenty-two seconds.

**Size and Growth.**—The relative size of the heart is slightly greater in infancy than in later life, it being smallest at about the seventh year. The average weight at the different periods of life is as follows:<sup>1</sup>

| Age.          | Ounces. | Grammes. | Ratio to body weight. |
|---------------|---------|----------|-----------------------|
| Birth .....   | 0.50    | 14       | 1 to 225              |
| 1 year .....  | 1.25    | 35       |                       |
| 2 years ..... | 1.87    | 53       |                       |
| 3 " .....     | 2.25    | 64       |                       |
| 7 " .....     | 2.80    | 80       | 1 to 280              |
| 14 " .....    | 5.84    | 166      | 1 to 222              |
| Adult .....   | 8.50    | 241      | 1 to 226              |

<sup>1</sup> The figures in infancy are from one hundred and fifty-five observations made in the New York Infant Asylum; the others are taken from Sahli.

The growth of the heart is rapid during the first three years, and nearly proportionate to that of the body. It is slowest from the third to the tenth year, and most rapid from the eleventh to the fifteenth year. At birth, the thickness of the right ventricle is very nearly the same as that of the left, the ratio being 6:7. The left ventricle, however, grows very much more rapidly than the right, so that at the end of the second year the ratio is 1:2, which is nearly that of the rest of childhood.

**Position of the Apex Beat.**—In the infant the heart is placed somewhat higher, and occupies a position a little nearer the horizontal than in the adult. This is partly due to the higher position of the diaphragm. The apex beat is therefore higher and farther to the left than in adult life. According to the observations of Wassilewski and Starck, whose combined examinations with reference to this point were made upon over 2,100 children, the apex beat is, as a rule, outside the mammary line until the fourth year; if it is less than one-third of an inch beyond the nipple, it can not be considered abnormal. From the fourth to the ninth year, the apex beat is in or near the mammary line. After the thirteenth year, under normal conditions, it is invariably within that line. During the first year the apex beat is usually found in the fourth intercostal space; from the first to the seventh year, it is found with about equal frequency in the fourth and the fifth spaces; after the seventh it is usually, and after the thirteenth year it is always, when normal, in the fifth space. The position of the apex beat may be considerably modified by severe deformities of the chest resulting from rickets, Pott's disease, or lateral curvature of the spine.

**Examination of the Heart.**—*Inspection.*—Bulging of the præcordia is a frequent and important sign of cardiac disease during childhood. The cardiac impulse is generally weaker than in the adult, and often it is difficult to locate the apex beat owing to the thick layer of adipose tissue covering the chest.

*Palpation.*—This is usually a much more satisfactory method than is inspection for determining the position of the apex beat. For this purpose the child should be in the sitting posture, with the body inclined slightly forward. Great displacement of the apex beat is always significant, and should lead one to suspect pleuritic effusion; lesser degrees of displacement to the left indicate hypertrophy, especially of the left ventricle; to the right, hypertrophy of the right ventricle.

*Percussion.*—This is best done by means of the percussion hammer. A light blow should be used, on account of the thinness and elasticity of the chest walls. In percussing the heart, changes in the percussion note are generally better appreciated if one proceeds from the lung toward the heart rather than in the opposite direction. The outline of the area of "relative" or "deep cardiac dulness," especially in small chil-

dren, is proportionately larger than in the adult. This may lead to the mistaken opinion that the heart is enlarged, when it is really of normal size. The upper boundary of this area is at the second interspace or the lower border of the second costal cartilage, at the left margin of the sternum; from this point the line of dulness extends in a curved direction outward and downward, the extreme left limit being at or slightly beyond the mammary line at the fourth interspace. On the right side the line of dulness extends downward from the second interspace in a slightly curved direction along the parasternal line. The lower border is indeterminable on account of the liver.

The area of "absolute" or "superficial cardiac dulness," or that part of the heart uncovered by the lung, resembles in shape the same area in the adult, but it is relatively larger.

*Auscultation.*—This is of little value unless the child is quiet. For an accurate diagnosis the stethoscope is indispensable, but auscultation should always be practised with the naked ear as well. The rhythm and rapidity of the child's heart action are much more easily disturbed than are the adult's, and such disturbances are consequently much less significant. The rapidity of the heart in infancy is ordinarily so great as to make it difficult to distinguish between diastolic and presystolic murmurs. Normally, the loudest sound is the first sound at the apex; the weakest sound is the second sound at the aortic orifice.

In consequence of the small size and the thin walls of the chest, all sounds, both normal and pathological, appear relatively louder than in the adult, and the area of diffusion is therefore much greater. Thus it is a frequent occurrence for murmurs to be heard all over the chest both in front and behind.

Reduplication of the heart sounds, in consequence of the valves of the two sides not closing exactly together, is not uncommon in children, and may be due simply to excitement. During the first four years of life nearly all the abnormal murmurs heard are systolic.

Accidental murmurs may be due to anæmia and other blood conditions, and, although not so common as in older patients, they are by no means rare even in infants.

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## CHAPTER II.

### CONGENITAL ANOMALIES OF THE HEART.

**Etiology.**—Of the causes of congenital cardiac disease little is definitely known. It occurs more often in first-born children than later ones; 16 of 50 cases being in first children (Still). It is often associated with other forms of imperfect development, notably of the brain, as



in Mongolian idiocy. An attempt has been made to connect cardiac malformations with syphilis. A syphilitic family history is very seldom found. But Warthin has lately brought forward additional reason for suspecting syphilis since he has found that some of these cases give a positive reaction to the Wassermann test. Further observations are needed on this point. There has not been adduced any evidence to show that rheumatism plays a part.

**Lesions.**—The congenital anomalies of the heart may be grouped under three general heads:

1. Malformations resulting from imperfect development of certain parts of the heart, most frequently one of the septa. Either the ventricular or the auricular septum may be affected, or that dividing the pulmonary artery from the aorta. Such failure in development perpetuates conditions which are normal in the early months of foetal life. There may also be atresia of any one of the orifices, absence of one or more of the valvular leaflets, or of any one of the large vessels.

2. The results of foetal endocarditis. The effects of this condition vary according to the time of its occurrence. It is almost invariably of the right side, most frequently affecting the pulmonic valves. Valvular disease in foetal life leads not only to hypertrophy and dilatation, but also interferes with the normal development of the heart by preventing the closure of the auricular or ventricular septum or the ductus arteriosus, these being kept open by way of compensation.

3. Persistence of foetal conditions, such as the foramen ovale or ductus arteriosus. This may be the result of valvular disease, as previously stated, or of some condition of the lungs, such as atelectasis.

In the following table are given the lesions found in two hundred and forty-two cases, which I have collected from medical literature:

*Frequency of the different lesions in 242 autopsies upon cases of congenital cardiac anomaly.*

|   |  |
|---|--|
| Defect in the ventricular septum . . . . .                        | 149 cases; the only lesion in 5 cases. |
| Defect in the auricular septum, or patent foramen ovale . . . . . | 126 " " " " " 9 "                      |
| Pulmonic stenosis or atresia . . . . .                            | 108 " " " " " 6 "                      |
| Patent ductus arteriosus . . . . .                                | 68 " " " " " 3 "                       |
| Abnormalities in the origin of the great vessels . . . . .        | 45 " " " " " 0 "                       |
| Pulmonic insufficiency . . . . .                                  | 17 " " " " " 0 "                       |
| Tricuspid insufficiency . . . . .                                 | 6 " " " " " 0 "                        |
| Tricuspid stenosis or atresia . . . . .                           | 3 " " " " " 0 "                        |
| Mitral insufficiency . . . . .                                    | 1 " " " " " 0 "                        |
| Mitral stenosis or atresia . . . . .                              | 6 " " " " " 0 "                        |
| Aortic insufficiency . . . . .                                    | 1 " " " " " 0 "                        |
| Aortic stenosis or atresia . . . . .                              | 6 " " " " " 0 "                        |
| Transposition of the heart . . . . .                              | 2 " " " " " 0 "                        |
| Ectocardia . . . . .  | 1 " " " " " 0 "                        |

*The most frequent associated lesions.*

|  |   |
|--|---|
| Pulmonic stenosis with defect in the ventricular septum..... | 92 cases; the only lesions in 20 cases. |
| Pulmonic stenosis, with defect in the auricular septum.....  | 52 " " " " " 8 "                        |
| Defects in both septa.....                                   | 82 " " " " " 17 "                       |
| Pulmonic stenosis with defects in both septa..               | 36 " " " " " 21 "                       |

From this table it will be seen that, in the great majority of cases, several lesions are present, the most frequent combinations being pulmonic stenosis with defective ventricular septum, pulmonic stenosis with defective auricular septum, the three lesions associated, or the first two with a patent ductus arteriosus. Stenosis of the isthmus of the aorta, although not noted in this series, is not a very uncommon lesion; the obstruction is in the arch of the aorta beyond the point where the large vessels are given off.

*Defect in the Ventricular Septum.*—This is the most frequent lesion in congenital cardiac disease, and in half the cases was associated with pulmonic stenosis. The defect is generally at the upper part of the septum (Fig. 98). It is usually from one-fourth to one-half an inch in diameter, but not infrequently there is a large defect, and the septum may be entirely absent, the heart then consisting of but three cavities—two auricles and one ventricle. If the auricular septum also is wanting, as may be the case, the heart has but two cavities. Frequently there are also abnormalities in the origin of the great vessels. The pulmonary artery and the aorta may be given off from the common ventricle, or the aorta may arise partly from one ventricle and partly from the other. If pulmonic stenosis or atresia is present, the opening in the ventricular septum is conservative, affording a channel for the passage of blood from the right to the left side of the heart.



FIG. 98.—CONGENITAL CARDIAC DISEASE. The left ventricle is shown with a defect in the ventricular septum, the opening being just beneath the aortic valve. (From a patient dying in the Babies' Hospital.)

*Patent Foramen Ovale, or Defect in the Auricular Septum.*—Although this is one of the most common congenital malformations, it is not one of the most important. It rarely occurs alone, but is frequently

found with pulmonic stenosis or a defect in the ventricular septum. Small oblique openings in the auricular septum—usually at the foramen ovale—are not infrequently met with in autopsies upon young infants, but they are of no importance. In pathological conditions the opening is from one-fourth to one inch in diameter, and there may be more than one opening. A defect in this septum is frequently secondary to pulmonic stenosis.

*Patent Ductus Arteriosus.*—As a solitary lesion this is rare, but it is frequently associated with pulmonic stenosis, usually with a defect in one or both septa. It is then one of the channels by which the blood may find its way to the lungs when the pulmonary orifice is obstructed. It is not a malformation, but simply the persistence of a foetal condition usually necessitated by other changes in the heart. But the direction of the blood current is the opposite of that which exists in foetal life.

*Pulmonic Stenosis.*—This is one of the most frequent and most important lesions. It may be due to foetal endocarditis, or to a malformation. If the former, there is usually stenosis; if the latter, there may be atresia. It is often a primary lesion, and when marked it is always accompanied by other changes, most frequently by a defect in one or both septa or by a patent ductus arteriosus. This is important, as being more constantly associated with cyanosis than is any other congenital lesion. Most of the children who live beyond six or seven years with cyanosis have this lesion, always accompanied by others of a compensatory character. The amount of obstruction varies from a slight narrowing of the orifice to complete atresia. The seat of obstruction may be at the pulmonic orifice, in the conus arteriosus, or in the pulmonary artery just beyond the valves. If there is atresia, the pulmonary artery is very small, and may be rudimentary.

*Pulmonic Insufficiency.*—This lesion is relatively rare. It is usually the result of foetal endocarditis, but there may be absence of the pulmonary valve. It is most frequently associated with a defect in the ventricular septum.

*Tricuspid, mitral, and aortic disease* are relatively infrequent and usually seen in cases with multiple defects. Atresia or stenosis is much more common than insufficiency.

*Abnormalities in the Origin of the Large Vessels.*—These are quite frequent; but, as will be seen from the table, they are always associated with other lesions. Three forms are seen: (1) Transposition of the large vessels—the pulmonary artery is given off from the left, and the aorta from the right, ventricle. (2) Both arteries arise from a common trunk. This is usually due to an incomplete development of the lower part of the septum dividing the two arteries. Usually the pulmonary artery appears to be a branch of the aorta. This condition is frequently associated with other abnormalities, often with so large a defect

in the ventricular septum that there is really but one ventricle. (3) The aorta has an abnormal origin, arising from the right ventricle, or partly from both ventricles. This also is associated with a large defect in the ventricular septum. When described as arising from both ventricles, the aorta is usually given off directly above the line of the septum.

An abnormality in the number of valvular segments is quite frequent, but seldom impairs the valve's function. In rare cases a valve is rudimentary, and it may be absent, generally at the pulmonic or tricuspid orifice. Absence of the right auricle and absence of the pericardium have been recorded; also opening of the pulmonary veins into the right auricle, and a single pulmonary artery. In one case in the series there was ectocardia, this being associated with a congenital fissure of the sternum. I once saw a very remarkable instance of congenital cardiac displacement; the heart was situated in the abdominal cavity. Its pulsations could be plainly seen and felt just above the umbilicus. There was a large umbilical hernia, a congenital defect of the abdominal walls, and undoubtedly also an opening in the diaphragm.<sup>1</sup>

*Transposition of the heart*, or true dextro-cardia, was recorded but twice in this series of cases. It was, however, simulated in several others, including one of my own, where the apex beat was to the right of the sternum. There was in this case great hypertrophy of the right ventricle with a rudimentary ventricular septum.

*Secondary Lesions.*—In congenital malformations the right heart is usually found hypertrophied, since there are present one or more of the foetal conditions in which the greater part of the work is thrown upon the right ventricle. Such hypertrophy is in most cases accompanied by some dilatation. Changes in the wall of the left heart alone are exceedingly rare. In four cases there was evidence of malignant endocarditis, which was the cause of death, all but one of these patients being adults.

*Symptoms.*—The symptoms of congenital cardiac disease are usually manifested soon after birth. Of 128 cases in which the time of the first symptoms was noted, they were congenital, or appeared during the first month, in 85; after one month and during the first year, in 18; from one to sixteen years, in 15; while in 10 no symptoms were observed until after puberty. Congenital cardiac disease is one of the causes, but not a frequent one, of death during the first days of life.

The most striking objective symptom is cyanosis. This is present in most of the severe cases; but, considering all varieties, cyanosis is more often absent than present, and it may be absent even with serious lesions. It may be slight and noticed only upon exertion, as upon coughing or crying, or it may be intense and constant, giving the skin a dark, leaden colour, and the mucous membrane of the mouth a rasp-

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<sup>1</sup> The Medical News, December 11, 1897.

berry hue. The view that cyanosis depends upon an admixture of arterial and venous blood is generally discredited. In the great majority of the cases at least, the explanation is a deficient oxidation of the blood in the lungs, owing to some interference with the pulmonary circulation. In sixty-three per cent of the cases with cyanosis in the series, there was found pulmonic stenosis or atresia, or a small pulmonary artery. Cyanosis is of much value in diagnosis, as in acquired cardiac disease it is rarely persistent. The degree of cyanosis and its constancy are of some importance in determining the gravity of the lesion, although cyanosis alone is not to be depended upon.

Another frequent symptom is the enlargement of the terminal phalanges known as clubbed or "drum-stick" fingers (Fig. 99) and



FIG. 99.—CLUBBING OF THE FINGERS IN CONGENITAL HEART DISEASE.  
(From a boy five years old.)

toes. This almost invariably accompanies cyanosis, and is generally proportionate to it. The enlargement, which usually involves all the phalanges, is probably due to venous obstruction. Occasionally there are seen dyspnoea, oedema of the face or lower extremities, dropsy of the serous cavities, and hæmorrhages, particularly hæmoptysis and epistaxis.

There is generally marked dyspnoea on exertion in the cases in which cyanosis is present; but in most of those without cyanosis there is no dyspnoea, and, in fact, no objective or subjective symptoms, even though the murmur may be very loud. The majority of the cyanotic cases are

undersized and develop slowly; in them the problem of nutrition is a difficult one.

In cases accompanied by cyanosis, or with obstruction to the pulmonary circulation, a polycythæmia is present. The increase in the number of red cells is generally proportionate to the cyanosis; the average of fifteen cases studied in my clinic by Dr. I. S. Wile was 7,495,000; the highest was 12,480,000. The hæmoglobin is usually correspondingly increased. In the series mentioned the average was 107 per cent, the highest being 130. The number of white cells is changed very slightly, if at all; the average in my cases was 10,200, which disproves the theory of blood concentration. The best explanation of the polycythæmia seems to be that it is compensatory, and that the blood hypertrophies like other tissues. The blood-forming organs are stimulated to greater activity by the demands of the tissues for oxygen. The quantity of blood remains the same, but the number of red cells and the hæmoglobin, and consequently the oxygen-carrying power, are very greatly increased. This in part compensates for the smaller amount of blood that can traverse the lungs and there become oxygenated.

**Diagnosis.**—The most important diagnostic features are cyanosis, the presence of a loud murmur, and signs of enlargement of the right heart.

Murmurs are present in fully nine-tenths of the cases, the most characteristic being a systolic murmur, loudest at the left border of the sternum in the second or third intercostal space, and widely diffused, often being audible all over the chest. The point of maximum intensity is important for diagnosis. In the great majority of cases only a single murmur is heard. A systolic murmur is usually due to pulmonic stenosis, deficient ventricular septum or aortic stenosis, very rarely to mitral or tricuspid regurgitation. Since these conditions are very often associated, it is difficult to tell upon which one the murmur depends.

A patent ductus arteriosus usually gives a prolonged, almost continuous, murmur with systolic intensification, which is loudest in the second or third left interspace. In a young child, a loud murmur at the base of the heart with cyanosis, almost always means congenital disease. A thrill is often present but it is not important for a diagnosis.

Enlargement of the right heart, chiefly from ventricular hypertrophy, is present in most of the cases.

A diagnosis of the precise nature of the malformation is very difficult, and in the great majority of cases only a probable diagnosis is possible. Nearly all the cases are complex, and the variety of combinations is very great. A study of the histories and autopsies of the cases in this series reveals many apparently contradictory facts. Loud murmurs are sometimes heard which are difficult to explain by the lesions, and murmurs may be absent when there is every reason from the post-mortem findings for expecting their presence. With reference to the other conditions, I

can not do better than give the more frequent clinical symptoms with the results of the autopsies in the series of cases which I have collected.

*A Systolic Murmur at the Base with Cyanosis.*—This was the most common combination met with, and was present in about one-third of the entire number. In over eighty per cent of the cases with these symptoms, pulmonic stenosis was found. The remainder were complicated cases of quite a wide variety. Pulmonic stenosis was usually associated with a defect in one of the cardiac septa, or a patent ductus arteriosus.

*A Systolic Murmur without Cyanosis.*—In this series of autopsies this was not a frequent combination, being noted but six times. It is usually dependent upon a defect in the ventricular septum without pulmonic stenosis. Clinically, however, this is more often seen, in fact it is one of the most common types. The murmur is generally loudest at the left margin of the sternum at the third space. There is a striking absence of all other symptoms. I have watched a number of such patients grow to maturity and go through serious attacks of illness without showing any symptoms referable to the heart.

*A Systolic Murmur at the Apex with Cyanosis.*—Of the six cases with this combination, all were examples of complex malformation, the most frequent lesions being a defect in the auricular septum, transposition of the great vessels, and patent ductus arteriosus.

*Cyanosis without murmurs* was noted fourteen times. It usually indicates either pulmonic atresia or the transposition or irregular origin of the great vessels, but is sometimes seen when lesions, such as usually give murmurs, are found at autopsy.

*Diastolic murmurs* were heard in two cases, and depended upon pulmonic insufficiency.

*Absence of both cyanosis and murmurs* was recorded in five cases. The lesions found were: atresia of the aorta, both arteries arising from the right ventricle, or defective septa.

The only cases, therefore, in which a fairly certain anatomical diagnosis can be made are those of pulmonic stenosis with a deficient ventricular septum.

*Diagnosis of Congenital from Acquired Disease.*—Congenital disease may be suspected if the patient is under two years of age; if there is no history of previous rheumatism; if the murmur is atypical in its location, character, or transmission; if there is a very loud murmur at the base or over the body of the heart, and if there is evidence of enlargement of the right heart. If cyanosis and clubbing of the fingers are present the diagnosis is almost certain.

Especially difficult are the cases without cyanosis seen in older children. But absence of hypertrophy of the left ventricle, continued absence of subjective symptoms, even with a very loud murmur, and a lesion

which does not increase, all point strongly to a congenital malformation.

*Diagnosis of Congenital from Accidental Murmurs.*—This is often a more difficult matter than to decide between congenital and acquired disease. From a murmur alone one should be very cautious in making a diagnosis of cardiac malformation in a very anæmic infant. Anæmic murmurs are systolic, usually basic, unaccompanied by enlargement of the heart; usually heard in the carotids, often in the subclavian arteries, but are seldom so loud as those due to malformations. In some cases it may be necessary to watch the progress of the case before deciding the question.

In children from three to ten years of age it is not uncommon to find about the level of the nipple at the left border of the sternum a soft systolic murmur best heard on lying down, which, as it usually disappears, must be considered functional. It is easily mistaken for a congenital murmur.

**Prognosis.**—Of 225 cases, 60 per cent were fatal before the end of the fifth year, and nearly one-half of these during the first two months; while sixteen per cent of the cases lived over sixteen years, and eight per cent over thirty years. The prognosis in cases without cyanosis is good; in many children the lesion has apparently little effect on the health or development. The prognosis is much worse in cases with cyanosis, and generally it is bad in proportion to the degree of cyanosis. The loudness of the murmur has no prognostic importance.

In the cases fatal soon after birth the usual lesions are large defects in the septa, transposition of the great vessels, or pulmonic atresia. In five of twenty-three cases dying thus early, the heart had but two cavities. Lesions which are compatible with the longest life are minor septum defects, and pulmonic stenosis which can be compensated for by hypertrophy of the right ventricle and in other ways. Many exceptional instances are recorded in which patients have lived a long time in spite of extreme deformities. One child with transposition of the pulmonary artery and aorta lived two and a half years. Tiedemann's case lived eleven years with a heart consisting of three cavities—two auricles and one ventricle—and with constant cyanosis. In three cases reported by Rokitansky, the patients lived over forty years with rudimentary auricular septa; cyanosis is not mentioned as being present. Gelpke's case had cyanosis, and lived twenty-seven years with rudimentary auricular and ventricular septa, and with no tricuspid opening. Patients with serious congenital cardiac lesions are especially susceptible to pulmonary diseases of all kinds and occasionally develop malignant endocarditis. Almost any acute illness may prove fatal.

**Treatment.**—These patients are prone to develop at times attacks resembling angina pectoris, which are best relieved by amylnitrite or by the use of morphine hypodermically. No treatment is of the



slightest avail in diminishing the amount of deformity or promoting the closure of any of the abnormal openings. All cases are to be treated symptomatically.

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### CHAPTER III.

#### *PERICARDITIS.*

**INFLAMMATION** of the pericardium is uncommon in infancy and early childhood, only two cases being seen in 726 consecutive autopsies at the New York Infant Asylum. But in later childhood pericarditis is more frequent and more serious than the same disease in adults.

Pericarditis is almost invariably a secondary disease, following (1) empyema or pleuro-pneumonia; (2) acute rheumatism; (3) acute infectious diseases, especially scarlet fever; (4) pyæmia; (5) tuberculosis; (6) local conditions. The relative importance of these causes differs with the age of the child. In infancy and early childhood nearly all the cases complicate disease of the lung or pleura, more frequently of the left side. After the fourth year rheumatism takes the first place as an etiological factor. Pericarditis is then generally associated with endocarditis, and may precede or follow the articular manifestations of rheumatism. Following scarlet fever, pericarditis often occurs in connection with nephritis or multiple joint inflammations. In typhoid fever also it is usually associated with pneumonia or joint lesions. Pyæmia may be a cause in the newly born, or it may occur in connection with disease of the bones or joints in older children; in both it is usually associated with similar lesions of other serous membranes. Tuberculous pericarditis is more frequent after the third year, and is generally secondary to pulmonary tuberculosis. Among the local causes may be mentioned traumatism, ulceration of a foreign body from the œsophagus into the pericardium, disease of the sternum, ribs, or vertebræ, and abscesses resulting from cheesy bronchial lymph nodes.

**Lesions.**—Pericardial transudations, or an increase in the normal pericardial fluid, are met with in many conditions in which there is a very marked degree of anæmia, general dropsy, or a weak heart, particularly of the right side. Generally from one and a half to two ounces of clear serum are found in the pericardial sac.

*Pneumococcus pericarditis* is always acute and closely resembles in its lesions the inflammation of the pleura due to the same cause. In the milder cases there is seen only a fibrinous exudate. In the more common and more severe forms the visceral and parietal pericardium is covered with a thick coating of fibrin and pus (compare pleuro-pneumonia), or more pus cells and serum may be poured out and the sac

contain fluid pus. The amount is usually small, one-half to one ounce, but it may be as much as a pint. When the inflammation is excited by other pyogenic organisms, the staphylococcus or the streptococcus, the lesions are similar to those just described.

In rheumatic pericarditis the inflammation may be a plastic one with a fibrino-cellular exudate (dry pericarditis) or sero-fibrinous (pericarditis with effusion). The inflammation generally begins at the base of the heart and affects both the visceral and parietal layers. The quantity of fluid present is usually small, not exceeding two or three ounces; exceptionally as much as a pint may be present. It may be clear or slightly turbid. More important than the pericarditis are the associated changes in the heart muscle. These are present in every severe case. To the myocarditis and consequent dilatation the most serious symptoms of pericarditis are due.

Purulent pericarditis may be set up by a foreign body ulcerating into the sac, by the rupture of a mediastinal abscess, or by general pyæmia. Under these circumstances the process may be purulent from the outset. Any of the pyogenic bacteria may be found.

External or mediastinal pericarditis is always associated with mediastinal pleurisy, and results in more or less extensive adhesions of the pericardial and pleural surfaces, with an increase in the connective tissue of the mediastinum. This is often a tuberculous process. When severe, it may cause compression of the large blood-vessels, but seldom in any other way produce symptoms. With this form there is usually inflammation of the internal layer of the pericardium as well. Only inflammation of the internal layer is ordinarily considered as pericarditis, the other form being preferably classed as mediastinitis.

Pericarditis with an effusion of blood is very rare in children. It may occur from the rupture of organised adhesions or in certain blood states such as purpura, and very rarely in tuberculosis.

With acute tuberculosis there is usually only a deposit of miliary tubercles, or there may be a small serous or sero-sanguinolent effusion. In chronic cases there may be a tuberculous inflammation with the formation of caseous nodules, new connective tissue, and extensive adhesions. This generally occurs in connection with pulmonary tuberculosis—sometimes with tuberculous peritonitis.

In any form of pericarditis complete recovery, so far as pathological conditions are concerned, is rare—if, indeed, it ever occurs. After a rheumatic pericarditis adhesions remain, which may be slight, but are often complete, causing entire obliteration of the pericardial sac. Such adhesions are followed by secondary changes. The growth and development of the heart are interfered with, and there may be sufficient pressure upon the coronary vessels to lead to degeneration of the muscular walls and chronic dilatation of the heart.

**Symptoms.**—A pericardial transudation, or dropsy of the pericardium, is very rarely large enough to make a diagnosis possible.

External pericarditis is seldom recognised during life, there being no symptoms except those of the pleurisy with which it is associated. Occasionally there may be heard, particularly if the inflammation is anterior, a pleuritic friction sound which is increased with the systole of the heart. The pulse may be weak during inspiration, and there may be an increased area of cardiac dulness. If the inflammation is chiefly posterior, it causes only the symptoms of mediastinitis, which is recognised principally by its pressure effects upon the great vessels. It may produce œdema of the face or of the lower extremities, ascites, enlargement of the liver and spleen, but rarely albuminuria. It is usually progressive, and lasts from a few months to two or three years, according to its cause.

Pericarditis in infancy is usually overlooked, not only on account of its rarity, but also from the obscurity of its symptoms. When pericarditis develops at the height of an attack of pneumonia, as it usually does, there may be no new symptoms, or at most only increased prostration with perhaps a more rapid or slightly irregular pulse. On auscultation, if practised early, one may get pericardial friction sounds; but these are masked by the pulmonary signs and in infants seldom made out. The most striking sign is that the cardiac sounds formerly distinct are now feeble and distant, at times almost inaudible. Later there may be increased dulness from pericardial effusion, or from dilatation. The physician should be on the watch for it in infants with pleuro-pneumonia, especially of the left side.

Rheumatic pericarditis affecting as it generally does older children is easier of recognition. Localised pain and tenderness are usually present and also a certain amount of embarrassment of the heart's action, manifested by præcordial distress, palpitation, or a tumultuous heart action with a rapid and at times an irregular pulse. There is often vomiting, dyspnoea, and a teasing, dry cough; there may be orthopnoea and some cyanosis. Sometimes there is delirium.

The earliest physical signs of pericarditis are friction sounds, which can generally be heard, though sometimes over only a small area, at the base of the heart. The sound is a double one; it is synchronous with the heart's movement, it is generally more circumscribed than an endocardial murmur and not so blowing in character. Very early there is an increase in cardiac dulness which may be considerable. It may extend as much as one and one-half inches beyond the right border of the sternum, and to the left one or two inches beyond the mammary line. (See Figs. 100 and 101.) It may be due to effusion or to dilatation with which effusion is easily and frequently confounded. In a case with early and rapidly developing dulness it is safe to assume that some dilatation is present. When there is considerable effusion the apex beat

is feeble and may be displaced upward. The cardiac sounds are diminished in intensity and may be almost inaudible. The area of dullness is triangular or pear-shaped with the base below. With large effusion there may also be dullness to the left of the spine behind. Rotch's sign of effusion, dullness to the right of the sternum in the fifth space, though often present is not entirely reliable.

In cases terminating fatally the progress of the disease is quite rapid, the entire duration being seldom longer than three or four weeks, and



FIG. 100.—PERICARDITIS WITH EFFUSION. Anterior view, showing moderate distention of the pericardium, especially to the right of the middle line; right border at A. Boy eight years old.



FIG. 101.—PERICARDITIS WITH EFFUSION. Same patient as Fig. 100, but taken four days later. Great distention of the pericardium; right border at B. Complete recovery by absorption.

it may be much less. Pneumonia often develops toward the close. When ending in recovery improvement is very slow and it may be two or three months before the patient is out of bed, and a much longer time before even a moderate degree of health is established.

**Prognosis.**—Acute pericarditis due to the pneumococcus in infancy almost invariably ends fatally and in older children this is the usual termination. Occasionally at the later age resolution may take place before pus forms, or the pyo-pericardium which ensues is successfully opened and drained. Purulent pericarditis from other causes is usually fatal. In rheumatic pericarditis the outlook for life is better, but this with its associated myocarditis is without doubt the gravest manifestation of rheumatism in early life. No complication is more to be dreaded, both on account of immediate and remote dangers. Of forty-eight cases of acute pericarditis reported by Still in which this supervened during endocarditis, forty proved fatal in the course of a few weeks. In patients who do not die from the disease the remote consequences by reason of adhesions and subsequent dilatation are very serious.

**Diagnosis.**—Pericarditis is recognised by knowing when to look for it—in infants with pneumonia, in older children with rheumatism. The

difficulties of diagnosis of dry pericarditis are very much greater in young children owing to the very rapid action of the heart. Dry pericarditis is recognised by the friction sounds, which are best heard over the base and are to be differentiated from endocardial murmurs. Pericarditis with effusion is to be diagnosticated from dilatation of the heart and from pleuritic effusions. From dilatation, the diagnosis is very difficult in childhood, but the recognition of small effusions is not essential, since the important condition is the accompanying dilatation. Large effusions may be mistaken for a sacculated empyema of the left side, in the latter, however, the heart is generally crowded to the right. When empyema and pericarditis coexist, it may be impossible to recognise the condition. The diagnosis between serous and purulent effusions can be made only by aspiration.

**Treatment.**—In an attack of acute pericarditis the patient should be kept in bed, absolutely quiet, and an ice-bag used over the heart. A layer of thin flannel should be placed beneath the bag. During the acute stage it should be applied constantly with perhaps a few hours' omission during the night. To be effective much attention to detail is necessary. Some children will not tolerate ice and for them dry heat may be substituted. It often mitigates the pain. Counter-irritation by mustard from time to time is useful, but blisters should not be employed in children. Leeching is much used in England, not so much in this country as its merits warrant. Four or five leeches are applied over the sternum or liver. The especial indications for leeches according to Still are cyanosis, marked dyspnoea, and a dilatation as shown by increase in the cardiac dulness. A rapid increase in dulness is to be regarded as mainly due to dilatation rather than effusion. Opium is, I think, of more value than any other drug. It has a steadying influence upon the excited heart, it relieves pain and also quiets the distressing cough. The form of administration is immaterial. The patient should be kept moderately under its influence throughout the active stage of the attack. Digitalis is sometimes useful, but must be used with caution. Alcohol is seldom indicated and has often done much harm in these cases. Strychnia and caffeine are much to be preferred when symptoms of heart failure are present. In the rheumatic form anti-rheumatic remedies are indicated, though it is still a question whether they accomplish very much after a severe pericarditis is once fairly under way. Either salicylate of soda or aspirin may be used. Serous effusions usually subside under simple tonic treatment. With very large serous effusions aspiration may relieve distressing symptoms, after which the rest of the fluid may undergo absorption. If the exploring needle shows the fluid to be purulent, incision and drainage should be practised as in empyema. The results of aspiration are exceedingly unfavourable. Of eighteen cases of aspiration of the pericardium collected by Keating, only four recovered. In puncturing the pericardium the point usually selected is a

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little to the left of the border of the sternum in the fifth intercostal space, the needle being directed upward and outward. In cases which do not end fatally a prolonged period of rest in bed is imperative on account of the dilatation.

#### CHRONIC PERICARDITIS WITH ADHESIONS.

This is not a very uncommon condition. It is usually general, but may be localised. The youngest case which has come under my observation was in a child sixteen months old, who died from acute bronchopneumonia. The adhesions were old and general, the pericardial sac being completely obliterated. Chronic adhesive pericarditis may follow single or repeated attacks of acute rheumatic pericarditis; it may be tuberculous. The pericardium may become very greatly thickened and its cavity obliterated; it may be adherent externally to the pleura, diaphragm, and chest wall. Other changes are usually present in the heart. It is often the seat of chronic myocarditis; the cavities are usually greatly dilated, and the heart walls much hypertrophied. Valvular lesions may be present.

Partial adhesions cause no symptoms by which they can be recognised, and even general adhesions sufficient to obliterate the pericardial sac may be found at autopsy when not suspected during life. This is one of the conditions in which, after it has led to considerable dilatation of the heart, sudden death sometimes occurs. Usually there is pallor, slight cyanosis, localised œdema of the chest and abdominal walls, and dyspnœa upon slight exertion. The liver and spleen are often enlarged and there may be ascites. These symptoms often lead to errors in diagnosis.

The heart is almost invariably much enlarged, chiefly from dilatation. On inspection, there may be bulging of the chest wall, with a diffused and often feeble or absent apex beat. The characteristic signs are a systolic retraction of the chest at or near the apex of the heart, sometimes at the tip of the sternum. This is due to the external pericardial adhesions, and is often better appreciated by palpation than by inspection. It is followed by a rapid rebound, associated with diastolic collapse of the jugular veins. Pulsus paradoxicus may also be present. Percussion shows an increase in the cardiac dulness in all directions. The position of the apex and the percussion outline of the heart do not change with the posture of the patient, and the cardiac dulness is but little affected by full inspiration. A systolic murmur is often present. The diagnosis of adherent pericardium always presents difficulties, but it can be made with tolerable certainty in a considerable proportion of cases. On account of the enlargement of the heart and the frequency of murmurs, it is usually mistaken for valvular disease. The prognosis is very bad. The lesion is a permanent one, and tends to increase. The treatment is symptomatic.

## CHAPTER IV.

*ENDOCARDITIS AND VALVULAR DISEASE OF THE HEART.*

ENDOCARDITIS may occur even in foetal life. At this period it usually affects the right side of the heart, and is one of the important causes of congenital malformations. In infancy, acute endocarditis is exceedingly rare, not a single instance being found in over one thousand autopsies upon children under three years of age of which I have records. From the third to the fifth year it is not so rare, and after five years is quite common.

The following table gives the age and sex in a series of cases of valvular disease observed by Dr. Crandall and myself:

| Age.        | 1<br>year. | 2<br>years. | 3<br>years. | 4<br>years. | 5<br>years. | 6<br>years. | 7<br>years. | 8<br>years. | 9<br>years. | 10<br>years. | 11<br>years. | 12<br>years. | 13<br>years. | 14<br>years. | Totals.    |
|-------------|------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|--------------|--------------|--------------|--------------|--------------|------------|
| Males . .   | ..         | 1           | 2           | 2           | 4           | 6           | 4           | 9           | 8           | 6            | 5            | 7            | 6            | 1            | 55, or 38% |
| Females . . | ..         | 1           | 3           | 5           | 7           | 9           | 10          | 3           | 11          | 12           | 14           | 4            | 2            | 3            | 90, " 62%  |
| Total.      | ..         | 2           | 5           | 7           | 11          | 15          | 14          | 12          | 19          | 18           | 19           | 11           | 8            | 4            | 145        |

The proportion of the sexes is very nearly the same as in my cases of rheumatism. Sturges, in 100 cases of chronic endocarditis, gives fifty-six per cent females and forty-four per cent males.

Endocarditis is usually spoken of as secondary to rheumatism; it is rather to be regarded as a manifestation, often the first, of that disease. Of 117 cases in my own series, ninety-three, or eighty per cent, gave a history of previous rheumatism. Of the 31 cases which at the first examination gave no history of rheumatism, 8 subsequently developed articular symptoms, and 2 chorea; so that nearly ninety per cent of this series of cases presented conclusive evidence of a rheumatic diathesis. Thirty per cent had chorea previously, or developed it while under observation. The proportion of rheumatic cases corresponds very closely with Cheadle's observations. In a series of 150 cases of valvular disease, Still found distinct evidences of rheumatism in 142.

Endocarditis may occur alone or with other manifestations of rheumatism. While frequently associated with acute articular rheumatism, in a much larger number it is seen with articular symptoms which are so slight as to be overlooked entirely or passed over as unimportant. It may occur with or follow chorea, tonsillitis, or torticollis, with or without articular symptoms. The proportion of rheumatic cases in which endocarditis occurs is much larger in children than in adults. In rare instances endocarditis is seen in the course of nearly all the infectious diseases, most frequently with scarlet fever, being often associated with pericarditis; but even in these conditions it is possible that it is some-

times rheumatic. The bacteriology of rheumatic endocarditis has not yet been determined with certainty.

**Lesions.**—In the great majority of cases endocarditis affects the mitral valve, and often only this. In 150 autopsies upon children dying of cardiac disease, Poynton found the mitral valve involved in 149, but in 76 of these the changes were not marked: in only 9 was there marked mitral stenosis. The aortic valve was affected in 51, but in only 9 was it seriously involved. Very striking was the frequency of pericarditis. Pericardial adhesions were present in 113 cases, and in 77 the adhesions were complete, i. e., the pericardial cavity was obliterated. These findings agree substantially with the observations of other English authorities, but in America the pericardial lesions are certainly not so prominent.

The pathological changes of acute endocarditis do not differ essentially in early life from those seen in adults. There is first an accumulation of bacteria upon the endocardium of the valves. These produce necrosis, which is followed by a clot formation, consisting chiefly of blood platelets and fibrin, in the meshes of which are leucocytes and a few red cells. The next change is a growth of new connective tissue cells and blood-vessels, which may be slight and superficial, but the rheumatic lesion usually extends deeply with an extensive proliferation of connective tissue which after a time undergoes contraction.

In the mildest forms of endocarditis it is possible for complete recovery to take place. In other cases there is left only a slight valvular thickening, not enough to interfere in any important way with function. In most patients, however, more marked changes are left. The valvular segments are swollen, adherent, somewhat shortened and consequently insufficient. Other changes in the heart usually accompany acute endocarditis. Dilatation is almost invariably present and is an important factor in producing insufficiency. In cases ending fatally there is very little hypertrophy: but if recovery occurs, hypertrophy develops and the lesion is compensated for in this way. A certain amount of myocarditis probably occurs in every severe case. It is most marked when pericarditis is also present. Emboli in children are rare. Subsequent attacks are exceedingly common and each one leaves the heart more seriously crippled.

Chronic inflammation may follow the first attack or more often occur after repeated attacks. The changes resulting from chronic endocarditis are practically identical with those seen in adult life and need not be described here. Emphasis, however, should be laid upon the fact that the younger the child the more rapid the progress of the disease.

**Symptoms.**—When endocarditis occurs as a primary disease, or when it is the only manifestation of rheumatism, it may begin abruptly with rather severe general symptoms—a temperature of 101° to 104° F., pros-



tration, exaggerated heart action, restlessness, and sometimes dyspnoea. More frequently, however, it begins much less acutely with only general malaise and slight fever, which often is not recognised without the thermometer. If the heart is not watched the diagnosis is not made and there may be no suspicion of the nature of the primary attack until some time afterward, when the existence of valvular disease is discovered. If, however, the heart is carefully and frequently examined there is heard, usually on the third or fourth day of the illness, a soft, blowing, systolic murmur at the apex.

Endocarditis occurring with rheumatism is by no means limited to those attacks with well-defined articular symptoms. It is very common and often severe when the articular symptoms are no more than stiffness, pain on motion, and slight swelling of the feet or ankles. There is no relation between the severity of these symptoms and the seriousness of the cardiac lesion. Occurring during chorea or after tonsillitis there may be nothing to call attention to the heart except sometimes an increased rapidity or irregularity of the pulse and possibly increased prostration; but frequently the cardiac condition is not suspected until the heart is examined.

Most of the cases of acute endocarditis seen in this country are of this mild type. Attacks of such severity as to produce death in the acute stage are relatively rare here, in marked contrast to the observations of English writers.

The usual duration of acute endocarditis is from two to four weeks, the general symptoms slowly subsiding and, if the case progresses favourably, the cardiac symptoms improve, but there is usually left behind a somewhat damaged heart because of valvular disease. In cases progressing unfavourably a fatal termination may come in the course of from two to six weeks owing usually to one of three causes or a combination of these: (1) The rapid development of dilatation accompanied by the usual signs of cardiac insufficiency; (2) pulmonary complications, generally pneumonia; (3) the supervention of acute pericarditis.

**Course of Chronic Valvular Disease.**—Chronic valvular disease follows one or more attacks of acute endocarditis, and may exist for months and sometimes for years, before it is recognised. Its course is usually divided into two periods, the first being that while compensation is present, and the second after compensation has failed. The duration of the stage of compensation is indefinite. The only subjective symptom that is of much diagnostic value is shortness of breath on exertion. Occasionally other symptoms are present, such as precordial pain, attacks of palpitation, headache, epistaxis, anæmia, loss of weight, and cough. These are rarely constant, but come on when the patient's general condition for any reason is below normal. As a rule, there is in young subjects a tendency to an increase in the disease, although this is often

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slow, and may be interrupted by long periods in which the process appears to be stationary. At such times the patients either have no symptoms, or suffer only from a slight amount of inconvenience on marked exertion.

Failure in compensation is generally brought about by one of the following causes: The most frequent is an intercurrent attack of rheumatism with a fresh endocarditis, which in a short time leads to a very great increase in the heart's disability. It may be due to additional work thrown upon the heart from excessive muscular exertion, or to the strain of a prolonged attack of some acute illness, especially one that is liable to produce changes in the heart muscle, such as typhoid, diphtheria, or scarlet fever. It is sometimes the increased work which is thrown upon the heart especially at the time of puberty, owing to the rapid growth of the body. It may result from any cause which seriously affects the patient's general nutrition, particularly when this is associated with marked anaemia.

The symptoms indicating failure of compensation are marked dyspnoea or orthopnoea and cough, sometimes accompanied by profuse expectoration, which may be bloody, and in rare cases there may be larger pulmonary hæmorrhages. With these may be associated other signs of pulmonary congestion and even pulmonary œdema. The obstruction to the systemic venous circulation leads to dropsy, which usually begins in the feet, sometimes in the face. There may be general anasarca and dropsy of the serous cavities, especially the peritonæum and pleura; also enlargement and functional disturbances of the liver, enlargement of the spleen, dyspeptic symptoms, and chronic congestion of the kidney, with scanty urine and albuminuria. There may be dilatation of the superficial veins and cyanosis; and there may be cerebral symptoms, such as headache, dizziness, and fainting attacks. The pulse is small and soft, and the heart's action rapid and irregular; the cardiac sounds are feeble and often indistinguishable, and it may be impossible to decide what murmurs, if any, are present.

It is rare to see all the symptoms of chronic progressive cardiac failure in children under ten years, but about the time of puberty they are common enough. The symptoms may increase in severity until death occurs, or they may be severe for a time and then nearly disappear, to return again after a longer or shorter interval.<sup>1</sup> Death may be due to sudden cardiac paralysis, to intercurrent nephritis, pneumonia, embolism, inflammation of the serous membranes, or to œdema of the lungs.

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<sup>1</sup> The course and termination of these cases of chronic valvular disease is well illustrated by the following history of a little girl who was under my observation for nine years: When first seen she was seven years old, and gave a history of cardiac symptoms for one year. There was then present a loud mitral regurgitant murmur, with considerable hypertrophy. There was general dropsy, and all the symptoms

*Physical Signs.*—Mitral murmurs are altogether the most common both in acute and chronic disease. Of 141 cases of valvular disease, in children under fourteen years, observed clinically, mitral murmurs were present in 135; in 131 the murmur of mitral insufficiency was heard, and in 99 this alone. In mitral insufficiency there is regurgitation of blood from the left ventricle into the left auricle during systole. There is heard a systolic murmur, synchronous with the apex impulse and with the first sound of the heart, which may wholly or in part replace the first sound. It is loudest at the apex, transmitted to the left, and usually heard at the inferior angle of the left scapula. In acute endocarditis the murmur is at first very soft and usually increases in intensity for several days. It may be represented by the syllables "whoo-ta" pronounced in a whisper. After attaining its maximum the murmur changes but little for some time. It may then diminish and eventually disappear entirely; but usually a murmur of moderate intensity remains. The only other important sign of acute endocarditis is enlargement of the heart which is almost entirely from dilatation. If the acute inflammation supervenes upon an old lesion, the previous murmur becomes louder and harsher. In chronic endocarditis the murmur is similar to that of acute endocarditis but generally louder and more widely diffused, and may be audible all over the chest. It is accompanied by an accentuation of the pulmonic second sound and by signs of hypertrophy, especially of the

pointed toward acute dilatation. Under treatment, the dropsy and other symptoms disappeared, and she went on comfortably for over a year. In her eighth and ninth years there were frequent attacks of subacute rheumatism, during which time the heart lesion steadily increased in severity. At twelve years there was an eruption of subcutaneous tendinous nodules, which remained for over two years. During this year there was heard for the first time a presystolic mitral murmur, accompanied by a very marked thrill, mitral stenosis having been gradually brought about by the slowly progressing endocarditis. This murmur gradually increased in intensity from that time, while the mitral regurgitant murmur became less distinct. The apex beat was then in the sixth space, two and a half inches to the left of the nipple. From the twelfth to the fifteenth year she grew very little in height or weight, and showed no signs of maturity, the cardiac symptoms being nearly stationary. In the fifteenth year she developed a marked enlargement of the liver and spleen with general dropsy and all the symptoms of cardiac insufficiency, these being the first symptoms of this character since she was seven years old. There was now heard for the first time an aortic regurgitant murmur in addition to the others formerly present. The symptoms disappeared under treatment in the course of a few months, but six months later returned with greater severity and were accompanied by albuminuria, the patient dying from heart failure in a few weeks. During the last exacerbation there was heard a double aortic as well as a double mitral murmur.

At autopsy the heart weighed fifteen ounces. There was a very great hypertrophy, especially of the right ventricle, which was as thick as the left. All the cavities were much dilated. The most important valvular lesion was mitral stenosis, the orifice not admitting the end of the little finger. The valves were the seat of calcareous deposits. The curtains of the aortic valve were thickened and adherent; there was also thickening of the pulmonic and tricuspid valves.

right heart. When both these signs are wanting, the existence of mitral insufficiency is somewhat doubtful, as a similar murmur may be functional or accidental. In the early stages of the disease and during compensation, the signs of hypertrophy predominate; in the later stages or with broken compensation, those of dilatation.

Mitral stenosis is relatively uncommon. It occurs after repeated attacks of rheumatism, with a slowly progressing endocarditis. It is usually associated with mitral regurgitation. With this lesion there is obstruction to the flow of blood from the left auricle into the left ventricle. It is mainly compensated for by hypertrophy of the right ventricle, but to a certain degree, also, by hypertrophy of the left auricle. The characteristic murmur of fully developed mitral stenosis is presystolic, prolonged, rough in character, and terminates abruptly with a sharp first sound of the heart. It is loudest at or just above the apex, but is audible over only a circumscribed area. Quite as constant and important for diagnosis is the presence of a "purring thrill," which is very distinct upon palpation, and terminates sharply as the apex strikes the chest wall. This murmur is not common in children and is heard only in cases in which cardiac disease has lasted several years.

With milder grades of mitral stenosis, or earlier in the course of the disease, there may be heard, shortly after the second sound, a murmur softer in quality and of short duration. It is usually audible above and to the inner side of the apex beat. In point of time this is often spoken of as the early diastolic murmur of mitral stenosis. It may be represented by the whispered syllables "whoo-ta-whoo," in which the first syllable is the mitral systolic murmur, which is somewhat prolonged; the second syllable is the second cardiac sound; the last is the early diastolic murmur, which is much shorter than the systolic murmur. The pulse of mitral stenosis is usually small.

Aortic lesions in children are much less common than mitral lesions, with which they are usually associated; they are seen in rather older patients. Aortic insufficiency is much more frequent than aortic stenosis. I have never seen it as the only lesion. It causes a regurgitation of blood from the aorta into the left ventricle during diastole. It is compensated for by dilatation and hypertrophy of the left ventricle. The signs of aortic insufficiency are a prolonged diastolic murmur, with or taking the place of the second sound of the heart, generally loudest at the left border of the sternum in the third space, and transmitted downward to the apex of the heart or the ensiform cartilage. This is invariably accompanied by signs of hypertrophy and dilatation of the left ventricle, which are usually marked. With great hypertrophy there is often bulging of the præcordium which may produce striking thoracic deformity. A characteristic symptom is the intense throbbing of the carotids, with the sudden distention followed by a complete collapse of

their walls, and the "water-hammer" pulse of Corrigan. A capillary pulse is often seen.

Aortic stenosis, unless congenital, is very rare in early life, and almost never occurs as the only lesion. Aortic stenosis is compensated for by hypertrophy of the left ventricle. It causes a systolic murmur, which is usually loudest at the right border of the sternum in the second space, and is transmitted upward, being distinct in the carotids. The second sound is generally weak and may be replaced by a diastolic murmur. A systolic thrill over the aortic area is usually present. Without the signs of hypertrophy of the left ventricle, a positive diagnosis should not be made.

Tricuspid insufficiency is usually secondary to disease of the left side of the heart, occurring in its late stages. It most frequently follows mitral insufficiency, where it is usually due to dilatation of the right ventricle without changes in the valves. It may be secondary to certain diseases of the lungs, such as emphysema, chronic interstitial pneumonia, or chronic pleurisy, and it may be due to congenital malformation. Tricuspid insufficiency gives a systolic murmur, loudest over the lower part of the sternum, but heard usually over a small area. It is associated with signs of dilatation of the right ventricle. The jugular veins stand out prominently, and often show systolic pulsation, especially upon the right side. The symptoms associated with tricuspid regurgitation are due to general systemic venous obstruction.

Tricuspid stenosis, pulmonic stenosis, and pulmonic insufficiency are practically unknown in childhood except as congenital lesions.

**Prognosis.**—The danger to life in acute endocarditis is not great unless it is accompanied by pericarditis; but when both are present the outlook is serious. Of 115 fatal cases reported by Poynton, thirty-five proved fatal in the primary attack. It is difficult during the active stage to foretell how serious will be the resulting damage to the heart. It is only by watching the progress of a case that one can decide. As a rule the younger the child the worse the prognosis.

Complete recovery from valvular disease is possible only when the lesions are very slight. Not many children die from chronic cardiac disease before reaching the age of ten or twelve years. Up to about the time of puberty many children do very well; then they begin to lose ground, and may fail rapidly. But more often it is a fresh endocarditis accompanying an intercurrent attack of rheumatism which marks the beginning of a downward course. The proportion of children who have serious cardiac lesions before the age of eight years and reach adult life in good condition is very small.

There are several features of cardiac disease in children, in consequence of which serious lesions tend to progress more rapidly than in adults. The muscular walls are less resistant, and hence dilatation oc-

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curs much more readily than in adult life. The heart must provide not only for constant needs, but for the growth of the body. If the patient's general nutrition is poor during the period of most rapid growth, this tells quickly and seriously upon the heart, and dilatation makes rapid progress. The demands made upon the heart at puberty are especially severe, by reason of the rapid growth of the body and the frequency of anæmia and malnutrition. There is always present the danger of rapid advances in the disease from intercurrent attacks of rheumatism, from which children are more likely to suffer than are older subjects. Extensive pericardial adhesions are frequent, and seriously handicap the heart, greatly increasing the tendency to dilatation. The effect upon the heart of poor food, unhygienic surroundings, and general malnutrition is much more marked than in adults.

These unfavourable conditions are in part offset by others in which the child has an advantage over the adult. Disease of the coronary arteries is very rare, and the valvular lesion which is most frequently met with—mitral insufficiency—is that which admits of the most complete compensation.

In making a prognosis in any given case, the amount of hypertrophy or dilatation which exists, and the presence or absence of pericardial adhesions are more important than the location or the special character of the murmur. The presence of valvular disease in childhood increases the danger from every acute disease, especially pertussis, diphtheria, pneumonia, and scarlet fever. The chances of recurring attacks of rheumatism must also be taken into account.

**Diagnosis.**—Valvular disease is to be particularly distinguished from conditions in which there are heard functional or accidental murmurs. According to my own experience the latter are quite common even in young children. Mistakes usually arise from attaching too much importance to the presence of murmurs, and too little to the changes in the walls and cavities of the heart, with which valvular disease is almost invariably associated. It is not always possible to decide whether a murmur is organic or functional until the patient has been for some time under observation and treatment, particularly when anæmia is present. The diagnostic points, so far as the murmurs are concerned, are mentioned in connection with accidental murmurs.

**Treatment.**—The first and altogether the most important indication for every case of recent endocarditis is to secure for the heart as complete rest as possible, not only during the period of active inflammation, but for several succeeding weeks. The reason for this is that some dilatation is always present and this very readily increases. With children, proper rest can be secured only by keeping them in bed; and, when possible, in a recumbent position. The duration of the period of rest after mild attacks of endocarditis should be at least six weeks, and after severe

attacks, three months. In these young patients changes in the walls of the heart take place very rapidly and the gravest consequences are liable to follow a neglect of these precautions. In old cases rest is indicated during every acute exacerbation; also whenever there is much dilatation and little hypertrophy, and whenever the signs of failing compensation are present. In these older cases rest is often impossible in the recumbent position; if secured at all, it must be obtained with the child in the sitting posture or at least propped up with pillows. Whether much can be accomplished by the administration of anti-rheumatic remedies after endocarditis has developed is very doubtful. Salicylates or aspirin and alkalis should, however, invariably be used with every fresh manifestation of rheumatism, to prevent, if possible, an increase in the cardiac inflammation. A child who is the subject of a chronic valvular disease should be constantly under a physician's observation. Irreparable harm often results from ignorant disregard of the simplest and most important rules of life for these patients.

Several distinct conditions may be present which call for quite different management. The essential points may be stated in a few words: For all recent cases and during all exacerbations, rest, complete and prolonged; for deformed valves with good heart walls and perfect compensation, fresh air, moderate exercise, and general tonics; for feeble heart walls, failing compensation and dilatation, rest and cardiac tonics.

During the stage of compensation, treatment directed especially to the heart is rarely necessary. The main purpose should be to maintain the patient's general nutrition at the highest possible point during the period of active growth. At the very least the patient should be carefully examined three or four times each year, in order that the physician may note the progress of the disease, and be able to direct the child's education, occupation, exercise, and surroundings so as to meet, as far as possible, the changing conditions. To this end, diet, sleep, study, and exercise should receive the most careful attention. If malnutrition and anæmia are allowed to go on unchecked until they become severe, the cardiac disease may make rapid strides, and as much harm be done in a few months as otherwise might not occur in years. The question of exercise and recreation is always a difficult one to settle. Often too little latitude is given, and the heart, like the voluntary muscles, loses its tone. Every form of exercise requiring a prolonged severe strain should be forbidden, particularly swimming and competitive games, like ball and tennis, and others requiring much running; but skating, rowing, horseback exercise, regulated gymnastics, and cycling on the level—all in moderation—may be allowed not only without harm, but with the greatest benefit; but any of these, used immoderately, may be productive of great injury. All exercise should be taken with regularity and system, the amount being carefully measured by the child's condition,

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and increased freedom allowed only after watching the effect. If the patient is a boy who must earn his own living, the physician should see to it that the occupation chosen is not one likely to make special demands upon the heart or to expose him unduly to conditions likely to induce rheumatism.

Special watchfulness is required at the time of puberty to prevent overpressure in schools, and the development of anaemia. The first symptoms of these conditions should be treated energetically, and if the heart seems to be overtaxed the child should be put to bed. Those who are specially liable to rheumatic attacks should, if possible, spend the winter and spring months in a warm, dry climate.

In the stage of failing compensation, the same general conditions are present as in adults, and they are to be managed in pretty much the same way. When such symptoms are first seen, prolonged rest in bed should be insisted upon as the thing most likely to restore the normal conditions. Digitalis and strophanthus are useful in children with about the same indications as in adults, viz., marked dilatation, dropsy, low arterial tension, and weak pulse. They may be used in doses of from five to ten drops of the tincture every four to six hours for a child of ten years. If there is much dilatation of the right side of the heart the same treatment is indicated as described in pericarditis. One should be cautious about using digitalis for an irregular and overacting heart, opium being decidedly preferable under these conditions. An overloaded venous circulation may be relieved by diuretics, by saline purgatives, or even by venesection. Iron and tonics generally are indicated, particularly strychnine and cod-liver oil.

#### MALIGNANT ENDOCARDITIS.

Malignant or ulcerative endocarditis is rare in childhood. The youngest cases I have found reported are one by Bond in an infant of two and a half months, and one by Harris in a boy four years old. In Bond's case the mitral valve was affected. It was due to the bacillus pyocyaneus. In Harris' case the right side of the heart was affected and the lesion was secondary to a congenital malformation. Of the cases reported in early life, most have been in children over ten years of age. Malignant endocarditis is rarely if ever primary. It may be seen in any infectious disease or septic process. In seventy-five per cent of the cases it is ingrafted upon a previous valvular disease. In my series of collected cases of congenital malformations of the heart, there were four deaths from malignant endocarditis, all but one, however, occurring in adult life. The bacteria most frequently concerned are the staphylococcus or streptococcus, next the pneumococcus, and rarely the gonococcus, the influenza or the pyocyaneus bacillus.

Malignant endocarditis presents a great variety of symptoms, often



making the diagnosis extremely difficult. There is generally a remittent type of fever, sometimes repeated rigors, sweating, low delirium, stupor or coma, and extreme prostration. There is often a fine petechial eruption. Usually there is a cardiac murmur, the location of which depends upon the seat of the disease; it is most frequently the murmur of mitral regurgitation. It is sometimes faint, and may be absent. From the emboli there may result hemiplegia, rapid swelling of the spleen, bloody urine or pneumonia. The disease lasts from three weeks to three months, death being the almost invariable termination. The most characteristic features of malignant endocarditis are the development of pyæmic or typhoid symptoms with a petechial eruption, in a patient who has previously had valvular disease. Blood cultures in most cases give positive results, though not always early in the disease.

The treatment is symptomatic. The use of vaccines has not met expectations; in the most acute cases no benefit has generally followed their administration, although in the more prolonged types they seem at times to have been of value.

#### MYOCARDITIS.

Disease of the muscular wall of the heart is rare in children, and of comparatively little importance, except in connection with acute endo- and pericarditis and the acute infectious diseases. It is almost invariably secondary to some infectious process. Aside from the rheumatic conditions already considered the diseases which furnish most of the cases are scarlet fever, diphtheria, and influenza. The most important local cause is pericarditis with adhesions.

**Lesions.**—In extra-uterine life, myocarditis as a rule affects chiefly the wall of the left ventricle, the papillary muscles, or the septum, but the entire organ is involved. The heart is of a grayish or yellowish-red colour, very soft, friable, and flabby, and there is frequently dilatation of the cavities.

Two varieties of myocarditis are described. In the parenchymatous form there is a degeneration of the muscle fibre which, according to Rombert, is most frequently albuminous, next fatty, and least frequently hyaline. There is a loss of the transverse striations, and there may be complete disintegration of the fibres. This process may be circumscribed, but it is usually diffuse. In the interstitial form the lesion usually occurs in small, circumscribed areas. There is an infiltration of round cells, chiefly mononuclear, between the muscular fibres of the heart. The process, when acute, may result in absorption or in the production of small abscesses. In chronic cases it may lead to the formation of areas of dense connective tissue resembling cicatrices, in the heart wall. Either the interstitial or the parenchymatous form may occur alone, but in most

of the acute cases they are combined. In addition, there is usually some degree of mural endocarditis and inflammation of the pericardium next to the heart wall. Dilatation frequently follows. Cardiac aneurism and even rupture have been known to occur in a child of six years (Hadden's case).

**Symptoms.**—In many cases in which advanced lesions have been found at autopsy there have been no symptoms appreciated during life. Careful examination of the heart, however, will usually show an alteration in the first cardiac sound, the muscular quality diminishing and the valvular quality increasing. This may go on even to a total disappearance of the muscular quality and only a flapping valvular sound may remain. The first and the second sounds are then almost alike. In such severe cases diastole is relatively short and the rhythm is much like that of foetal life. A systolic murmur due to dilatation of the auriculo-ventricular ring, or to imperfect action of the papillary muscles, may be heard at the apex. The heart is usually slightly dilated, but may be excessively so. Its action is generally increased in rapidity and may be irregular; a slow heart, 50 to 70, with feeble, valvular sounds is less frequent but very characteristic. The apex beat is diminished in intensity and the pulse is soft and weak. The blood pressure is low, frequently 60 mm. or even less. Other symptoms may be present that are dependent upon feeble heart action—pallor, dyspnoea, slight cyanosis, and attacks of syncope. Less frequently there may be dropsy of the feet or the serous cavities, and scanty urine, sometimes containing albumin. Death may occur suddenly from cardiac paralysis or gradually from circulatory failure. Recovery may take place after alarming symptoms have been present, these slowly abating. It may be many weeks before the normal cardiac sounds are heard.

**Treatment.**—This is mainly symptomatic. After severe attacks of those infectious diseases in which myocarditis is liable to occur, and at any time when it is suspected, patients should be kept recumbent for several weeks, and special care exercised to prevent any sudden exertion, as death has resulted from so slight a thing as suddenly sitting up in bed. Once definite symptoms have developed, absolute rest is imperative. Much more is to be expected from complete rest than from drugs, which as often employed may do positive harm. Digitalis should be used with caution, and never in large doses. In some cases with symptoms indicating imminent heart failure rather striking benefit has followed the use of morphine hypodermically.

#### ACCIDENTAL MURMURS.

In a paragraph upon the diagnosis of congenital cardiac disease, reference has already been made to a type of murmur frequently heard

in children and which may be confounded with a murmur due to organic disease.

Accidental murmurs may also be heard in cases of marked **anæmia**. These are not rare even in infancy. They may be confounded with organic murmurs either from congenital malformations or from acquired disease. In any anæmic infant, as well as older child, one should hesitate to make a diagnosis either of congenital or acquired organic disease, from the mere presence of a murmur.

An anæmic murmur is usually systolic, generally but not always loudest at the base of the heart, audible in the carotids, often in the subclavians, and occasionally over any large artery. The murmur varies from day to day, and sometimes it is altered by changing the position of the patient. It may be loud enough to be heard over a great part of the chest in front, and even behind. There is frequently present a venous hum in the neck. There are no signs of hypertrophy, nor is there the accentuated second sound so characteristic of mitral disease. The pulse is not usually strong. Anæmic murmurs diminish in intensity and ultimately disappear with improvement in the general condition of the patient.

#### FUNCTIONAL DISORDERS OF THE HEART.

Disturbances in the heart's action unconnected with organic disease, are not very common in young children; but after the seventh year they increase in frequency up to the time of puberty. One of the most important causes is indigestion; another is overpressure in schools, or anything else leading to nervous exhaustion. In these circumstances it is usually associated with other mental or psychical disturbances. An important predisposing cause is the demand made upon the heart by the rapid growth of the body about the time of puberty, particularly when this is associated with anæmia. In some of the cases there is a definite exciting cause, such as fright or great excitement, and it may be due to the excessive use of tea, coffee, or tobacco, especially in the form of cigarette-smoking. In a few instances it has been traced to masturbation. It may follow any acute disease, such as typhoid fever, malaria, or one of the exanthemata, and occasionally it occurs in the course of these diseases, or with bronchitis or pneumonia.

**Symptoms.**—The usual manifestations are attacks of palpitation; less frequently there is tachycardia or bradycardia. The majority of children complain more with functional disturbances than with organic disease, certainly while the latter is accompanied by compensation. Attacks of palpitation occur in paroxysms. In the severe form there is usually a sense of oppression in the region of the heart, with some dyspnœa, or even orthopnœa. The pulse is usually rapid, from 120 to 130, and is irregular both as to force and rhythm. The carotids pulsate strongly.

The apex impulse is felt over an increased area, the heart sounds are usually strong but irregular, and sometimes a slight murmur is heard. The face is pale or flushed. There may be headache, vertigo, spots before the eyes, and noises in the ears. Sometimes there is slight cyanosis with cold hands and feet, and general perspiration. The frequency of these attacks depends upon the nature of the exciting cause. Their duration is from a few minutes to several hours.

**Diagnosis.**—Functional disorders are differentiated from organic cardiac disease only by careful and repeated examinations of the heart. In the diagnosis of functional disturbance especial importance is to be attached to a neurotic or neurasthenic condition of the patient, to the presence of some adequate exciting cause, the absence of evidence of enlargement of the heart, and the fact that the pulmonic second sound is not increased.

**Prognosis.**—This in most cases is favourable, for with the removal of the cause, with improvement in the patient's general condition, with the growth of the body, and in girls with the establishment of menstruation, the attacks usually disappear.

**Treatment.**—The curative treatment is to be directed toward the cause. When no special cause can be discovered a general tonic plan of treatment should be adopted, with careful regulation of the patient's diet, exercise, and mode of life. All stimulating food, tea, coffee, and tobacco should be prohibited. Anæmia should receive its appropriate remedies. The hours of sleep and study, and the amount and character of exercise allowed, should be carefully regulated. Between attacks no treatment of the heart is necessary. During attacks bromides or valerian may be useful.

#### DISEASES OF THE BLOOD-VESSELS.

**Abnormally Small Arteries** (*Arterial hypoplasia*).—This condition is not a very common one, but it has attracted a good deal of attention, having been studied especially by Virchow. The only thing which is abnormal in the circulatory system may be that the aorta, and sometimes all the large vessels are only two-thirds or three-fourths their usual calibre, or even less. This may interfere seriously with the growth and development of the body, especially of the genital organs, although this result is not a constant one. The condition is found occasionally in cases of chlorosis, and in the congenital cases it may be the chief cause. There is sometimes associated a certain amount of hypertrophy of the heart. The other symptoms are anæmia, and sometimes an imperfect development of the body. A positive diagnosis during life is impossible.

**Aneurism and Atheroma.**—In early life chronic disease of the blood-vessels is exceedingly rare, yet a sufficient number of observations have been recorded to show that even young children are not exempt from this

form of disease. Sanné<sup>1</sup> records the youngest case, which occurred in a fœtus born at about the eighth month, in whose body there was found a large aneurism of the abdominal aorta just below the origin of the renal arteries. Le Boutillier<sup>2</sup> has collected seven cases of thoracic aneurism in children under ten years; the arch of the aorta was the usual seat.

Probably the most important etiological factor, as in adult life, is syphilis, but in only a few of the cases reported was the evidence of syphilis conclusive. In two cases there was general tuberculosis. In at least two cases whooping-cough appeared to act as a contributing cause. Aneurism may also be due to some local condition, such as an erosion from a bony growth, an abscess in the neighbourhood, or to embolism. The symptoms and course of aneurism in young children do not differ essentially from those of the disease as seen in adults.

In addition to the cases of aneurism referred to above, I have found reports of seven cases of atheroma in very young subjects. In Sanné's case the patient was but two years old, and patches of atheromatous degeneration were found in several places in the aorta. In Hawkins' case, eleven years old, there was found extensive atheromatous disease of the aorta, subclavian and carotid arteries. In Filatoff's case, atheromatous degeneration affected the arteries at the base of the brain, causing death from cerebral hæmorrhage. It is interesting to note that in this patient, who was only eleven years old, there was also present chronic diffuse nephritis with contracted kidneys. A similar condition of the kidneys and arteries was observed by Dickinson in a girl of six years.

**Embolism and Thrombosis.**—Embolism is very rare in early life, even with acute endocarditis. The emboli are usually swept into the circulation from vegetations upon the valves of the heart. The symptoms which they produce will depend upon the nature of the emboli and the vessels occluded by them. If they lodge in the brain they may cause paralysis or convulsions; if in the spleen, pain and swelling of this organ; if in the kidneys, pain, tenderness, and sometimes hæmaturia; if in the lungs, cough, sometimes accompanied by hæmoptysis and occasionally by a sharp thoracic pain. If the emboli are infectious, they may give rise to abscesses. The pathological results following embolism are similar to those which are seen in adults.

The most frequent form of thrombosis, that occurring in the sinuses of the brain, is discussed in connection with Diseases of the Nervous System. Cardiac thrombi, especially of the right side of the heart, are not infrequently found in patients dying from heart disease, pneumonia, and occasionally also from other acute inflammatory processes

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<sup>1</sup> Sanné, *Revue Mensuelle des Maladies des l'Enfance*, vol. v, p. 56.

<sup>2</sup> *American Journal of the Medical Sciences*, May, 1906. In these articles will be found references to most of the reported cases.

and acute infectious diseases, particularly diphtheria. These thrombi are in most cases produced during the last few hours of life, or just at the time of death, and are of no clinical importance. They frequently extend from the heart into the large blood-vessels, particularly the pulmonary artery. Thrombosis occasionally occurs in all the large vascular trunks in childhood as well as in adult life.

*Thrombosis of the Internal Jugular Vein.*—Pasteur<sup>1</sup> reports a case in a child two and a half years old, in which the middle of the vein was filled with an organised thrombus, and the lower portion obliterated and reduced to a fibrous cord. The symptoms were swelling, œdema, and cyanosis of the face, and dilatation of the facial vein, but not of the external jugular. There were clubbing of the fingers and œdema of the feet, but not of the arm. The heart was found to be dilated and hypertrophied, but was not the seat of valvular disease. The symptoms had existed since an attack of pneumonia, eighteen months before death.

*Thrombosis of the Vena Cava.*—Quite a number of cases are on record where this has occurred as the result of pressure from large abdominal tumours; it has followed new growths of the kidney and large masses of tuberculous lymph nodes. Neurutter and Salmon have recorded a case of thrombosis, apparently of marantic origin, in a child seven years old. The thrombus filled the vena cava, and extended to the origin of the hepatic veins and into both femorals. Death occurred from tuberculosis. In Scudder's case (seventeen years old) there was apparently obliteration (probably congenital) of the inferior vena cava; there was an extensive varicose condition of all the abdominal veins. The symptoms of thrombosis of the vena cava are swelling and œdema of the feet—sometimes of the abdominal walls and the groin—and very great dilatation of the superficial abdominal veins.

*Thrombosis of the Aorta.*—A case has been reported by Leopold in a newly-born child which was delivered by version. The thrombus was of recent origin, and filled the lower aorta, extending into the femoral artery. A case of thrombosis of the aorta occurring in a girl of thirteen years has been reported by Wallis. The aorta was very narrow, and probably the seat of syphilitic disease. The thrombus extended from the origin of the renal arteries to the celiac axis.

*Thrombosis in Infectious Diseases.*—There is occasionally seen in typhoid fever, but more frequently in diphtheria, thrombosis of some of the large venous trunks, usually of one of the lower extremities. The symptoms are pain, localised swelling, and partial paralysis. If the artery is affected, there may be gangrene.

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<sup>1</sup> Lancet, February 11, 1888.

SECTION VI.  
DISEASES OF THE URO-GENITAL SYSTEM.

CHAPTER I.

*THE URINE IN INFANCY AND CHILDHOOD.*

WHILE a study of the urine is of much less importance in early life than of the symptoms referable either to the digestive or respiratory system, it is deserving of much more attention than it has generally received. In infancy especially it is attended with some difficulty, owing to the fact that it is by no means an easy matter to secure readily specimens for examination.

**Methods of Collecting Urine.**—In male infants this may be done by placing the penis in the neck of a small bottle, which lies between the thighs, and is secured in position by pieces of tape passing over the hips and beneath the perinæum. The urine of female infants can sometimes be collected in a similar way by placing a small cup or a large-mouthed bottle over the vulva and holding it in place by the napkin or by pieces of adhesive plaster. A plan often successful is to put the infant upon a chamber after a long sleep. It should be done at the instant of waking or the child may be wakened for the purpose. A cold hand over the bladder facilitates matters. A small amount, sufficient to test for albumin, may often be obtained by placing absorbent cotton over the vulva or penis. The most certain of all means, however, is catheterisation, which, however, should not be resorted to unless absolutely necessary. A soft-rubber catheter, size 6 or 7, American scale (9 or 11 French), should be used for infants.

**Daily Quantity.**—This is relatively much larger in infants than in older children and in adults, on account of the large amount of water taken with the food. The quantity fluctuates widely from day to day, according to the amount of fluid food taken and the activity of the skin and bowels. The figures on the opposite page are the averages obtained by combining the results of the investigations of Schabanowa, Cruse, Camerer, Pollak, Martin-Ruge, Berti, Schiff, and Herter.

**Frequency of Micturition.**—This is greatest in young infants, and diminishes steadily as age advances. In infancy, during the waking hours, the urine is passed very frequently, often two or three times an

*Average Daily Quantity of Urine in Health.*

| AGE.                               | Grammes.      | Ounces. |
|------------------------------------|---------------|---------|
| First twenty-four hours . . . . .  | 0 to 60       | 0 to 2  |
| Second twenty-four hours . . . . . | 10 " 90       | ½ " 3   |
| Three to six days . . . . .        | 90 " 250      | 3 " 8   |
| Seven days to two months . . . . . | 150 " 400     | 5 " 13  |
| Two to six months . . . . .        | 210 " 500     | 7 " 16  |
| Six months to two years . . . . .  | 250 " 600     | 8 " 20  |
| Two to five years . . . . .        | 500 " 800     | 16 " 26 |
| Five to eight years . . . . .      | 600 " 1,200   | 20 " 40 |
| Eight to fourteen years . . . . .  | 1,000 " 1,500 | 32 " 48 |

hour, while during sleep it is retained from two to six hours. By the third year the urine may be held during sleep for eight or nine hours, and at other times for two or three hours. Such control of the sphincter of the bladder is often obtained at two years, and sometimes even at an earlier period. From slight nervous disturbances or minor ailments of any kind, this control is impaired, and the water may be passed by children of four or five years with the frequency seen in infants.

**Physical Character and Composition.**—The urine of the newly born is usually highly coloured. During later infancy it is pale and frequently turbid, even when practically normal, owing to the presence of mucus; this turbidity often no amount of filtration will entirely remove. Less frequently, turbidity depends upon urates. The urine of the first few days of life often shows a deposit of urates or uric acid in the form of a pink or reddish-yellow stain upon the napkin. The reaction of the urine at this time is usually strongly acid, but throughout the rest of infancy it is faintly acid or neutral.

The specific gravity is higher during the first two days than at any time in infancy on account of the scanty supply of fluid taken; it is usually lowest from the third to the sixth day, but from this time it rises steadily until puberty is reached. The specific gravity varies with the quantity. From the writers already referred to, the following figures are taken:

|                                    | Specific gravity. |
|------------------------------------|-------------------|
| First to third day . . . . .       | 1.010 to 1.012    |
| Fourth to tenth day . . . . .      | 1.004 " 1.008     |
| Tenth day to sixth month . . . . . | 1.004 " 1.010     |
| Six months to two years . . . . .  | 1.006 " 1.012     |
| Two to eight years . . . . .       | 1.008 " 1.016     |
| Eight to fourteen years . . . . .  | 1.012 " 1.020     |

Microscopically, the urine of the newly born shows the presence of many squamous epithelial cells, mucus, granular matter, and crystals of uric acid and amorphous or crystalline urates. It is not uncommon to



find hyaline and even granular casts. Martin-Ruge found hyaline casts in the urine of fourteen out of twenty-four healthy nursing infants examined during the first week. Granular casts were much less frequent. The microscopical appearances of the normal urine of later infancy and childhood present no peculiarities.

The inorganic salts (phosphates, chlorides, sulphates) are all present in the urine of the newly born, but in relatively small quantities, increasing as age advances. The colouring matters are also less abundant.

Albumin is often present in the urine during the first days, but usually in small amount. Cruse found it twenty-eight times in ninety observations upon healthy infants; usually the quantity was small, amounting to traces only, but in two cases it was quite large upon the second day. These observations are confirmed by the investigations of Martin-Ruge, and also of Pollak.

Sugar is frequently found in the urine of healthy infants during the first two months. It may be made to appear in the urine of healthy infants by simply increasing the quantity ingested. The different sugars vary as regards the amount which can be taken before it is thus eliminated. According to Grósz, lactose appears if the quantity is increased to three or four grammes per kilo. of body weight; glucose, only when five grammes, and maltose, not until seven and seven-tenths grammes per kilo. are given.

#### CYCLIC OR ORTHOSTATIC ALBUMINURIA.

**Etiology.**—This condition, although a rare one in young children, is occasionally seen between the ages of ten and sixteen years. I shall not in this connection include cases sometimes classed as febrile albuminuria, in which there is usually present the condition described as acute degeneration of the kidneys.

The causes of orthostatic albuminuria, and the circumstances in which it has been observed, are many and varied. It is much more common in males than in females. In certain cases albuminuria is distinctly traceable to cold bathing; in others, to fatigue following excessive muscular exercise; in still others, to dyspeptic conditions. It may be associated with a diet rich in nitrogenous food. Sometimes none of these conditions exist, and there is simply the occasional presence of albumin in the urine.

The theory which most satisfactorily explains this condition is that the most important factor is a mechanical one—that the albuminuria is due to the upright position. The vascular pressure in the kidney may be increased by deformities of the spine.

**Symptoms.**—Many of the patients exhibiting cyclic or periodic albuminuria are well nourished, and have no other signs of disease; others show dyspeptic symptoms, and are anæmic and poorly nourished, suffer-

ing from headaches and other neuroses. The amount of albumin is commonly small. In many patients albuminuria is regularly cyclic in character, albumin being absent in the urine passed during the night or early morning, but present during the day. In a case reported by Tiemann, the morning urine showed no trace of albumin in seventy-eight of eighty-four examinations. At noon albumin was present in ninety-eight of one hundred and thirteen examinations. It is not infrequently associated with temporary glycosuria. As a rule, casts are absent, although it is not uncommon to find a few hyaline casts, and occasionally granular casts are also present. But dropsy, cardiac hypertrophy, a pulse of high tension, retinal changes, and the characteristic symptoms of nephritis are absent.

Too much stress is certainly laid by Pavy and many other writers upon the fact that the albumin is found in the urine only at certain times in the day. This is not peculiar to functional albuminuria, as the same thing occurs in many cases of chronic nephritis, especially in the early stages, when the amount of albumin present is small. All these cases must be carefully watched for a long time and many observations made, before nephritis can positively be excluded.

**Prognosis.**—The prognosis in purely functional albuminuria is good. But many patients who for a considerable time were thought to have only functional albuminuria have ultimately developed nephritis. A favourable prognosis is therefore possible only after long observation.

**Treatment.**—This is to be directed toward the patient's general condition. Dyspeptic symptoms must be relieved, the patient's mode of life regulated, only moderate exercise allowed, and a simple diet given. If the urine is of high specific gravity, and contains oxalate of lime crystals, alkalies and mineral waters should be given in addition. Iron is indicated if there is anæmia.

## HÆMATURIA.

Hæmaturia is characterised by the presence of red blood-cells in the urine, and is to be distinguished from hæmoglobinuria where only blood pigment is present.

Hæmaturia may result from local or general causes. In infancy it may be due to new growths of the kidney. Such hæmorrhages, though rare, may be abundant, and may be seen early. Hæmaturia may occur also as a symptom of acute nephritis, especially that complicating scarlet fever, or it may result from the irritation of a calculus in the kidney, the ureter, or the bladder. In rare instances its cause is a new growth of the bladder, and it may be due to traumatism. It may sometimes be produced by the irritation of a highly concentrated urine, owing to the fact that too little fluid is taken. I saw a marked example of this in an

infant eight months old, where no other explanation could be found. I once saw hæmaturia following uric-acid infarctions in the newly born. It may also occur at this time as one of the symptoms of sepsis. Among the general causes the most important are: the hæmorrhagic disease of the newly born; the blood dyscrasiæ, such as scurvy, purpura, and hæmophilia; and infectious diseases, particularly typhoid, scarlet fever, influenza, and malaria. In most of these cases the amount of blood passed is small. When it is large it may appear in the urine as clear blood, or as clots, or it may impart simply a reddish or smoky colour to the urine. The colour, however, is not so reliable as a microscopical examination.

Large hæmorrhages are much more likely to come from the kidneys than from the bladder. The presence of blood casts from the renal tubules, or larger ones from the ureter, are conclusive evidence of the renal origin of the hæmorrhage.

The treatment of hæmaturia should be directed to the cause upon which it depends. In infancy scurvy especially should not be overlooked.

#### HÆMOGLOBINURIA.

In this condition blood pigment appears in the urine in large quantity, but red blood-cells are very few in number, or are absent altogether. In severe cases the urine may be almost black. There is commonly a small amount of albumin. This condition may be recognised by the appearance of granules of pigment under the microscope, or by Heller's test; the most conclusive means of diagnosis, however, is by the spectroscope.

Epidemic hæmoglobinuria (Winckel's disease) has already been described in the chapter on Diseases of the Newly Born. Hæmoglobinuria may be due to certain poisons, as carbolic acid or chlorate of potash, or to certain infectious diseases, as scarlet fever, typhoid fever, malaria, syphilis, or erysipelas.

Paroxysmal hæmoglobinuria occurs in childhood, although it is an exceedingly rare condition. A typical case in a child of four and a half years has been reported by Mackenzie. This was a delicate child of syphilitic parents; the hæmoglobinuria was preceded by fever and chills, without any other evidence of the presence of malaria. In certain children it follows exposure to cold or chilling of the surface of the body. The treatment of this condition is very unsatisfactory, but susceptible individuals should reside in a warm climate. For further description text-books on general medicine should be consulted.

#### PYURIA.

Pus in the urine may exist as an acute or a chronic condition. In either case, in a child, it is much more likely to come from the pelvis of

the kidney than from any other source. It may, however, come from any part of the genito-urinary tract—the kidney or its pelvis, the ureters, the bladder, the urethra, or the vagina. Sometimes it comes from an outside source, as when an abscess from perinephritis, appendicitis, or caries of the spine opens into the urinary tract.

Coming from the pelvis of the kidney, pus may indicate, if the condition is an acute one, pyelitis, pyelo-nephritis, or pyonephrosis; if it is chronic, it points to renal tuberculosis or calculus. The amount of pus in any of these conditions may be quite large. The urine is turbid and usually acid in reaction. It contains many epithelial cells of the transitional variety. A urine containing much pus is always albuminous. It is rare that pus comes from the ureters except in connection with congenital malformations or the impaction of calculi. Pus from the bladder is not usually in large quantity, and may be mixed with mucus. The urine may be alkaline or acid in reaction; there may be associated the symptoms of vesical irritation or of cystitis. Pus from the lower genital tract is rare in children, and its causes may often be recognised by a local examination. When the cause of pyuria is the opening of an abscess into the urinary tract there is generally a sudden appearance of pus in large amount. The pyuria is usually in such cases of short duration, possibly only a few days, and it may disappear quite rapidly.

The nature of the infection can be determined only by cultures made from a catheterised specimen. This information is of considerable aid both in diagnosis and prognosis.

The treatment of pyuria depends altogether upon its cause. Improvement in the symptoms sometimes follows the use of hexamethylenamine (urotropin), which may be given in doses of from two to five grains three times a day to a child of five years.

#### INDICANURIA.

Indicanuria is a condition characterised by the presence of indican in the urine. Indican (indoxyl-potassium sulphate) is derived from indol, which is formed in the intestine by the agency of bacteria from the excessive putrefaction of protein. It may also be produced in other parts of the body where putrefactive processes are going on, as in extensive suppuration without drainage, in pulmonary cavities, empyema, etc. Indican is only one of the ethereal sulphates produced in the manner above indicated, and when other conditions like those mentioned are excluded it may be taken as an index of the amount of putrefaction going on in the intestine.

Indicanuria is most frequently a symptom either of acute or chronic intestinal disease. It is important as being a guide by which we may

estimate the other symptoms in these conditions, and the effects of treatment. While a trace of indican is frequently present in health, a strong indican reaction is always to be considered abnormal in a child. The indications are to diminish intestinal putrefaction. The treatment is mainly dietetic. Indicanuria is usually increased by a meat diet and diminished by a milk diet. Other measures are referred to in the treatment of chronic intestinal indigestion.

#### ACETONURIA—DIACETONURIA, ETC.

Acetone exists in small quantities in the urine of healthy children. It is also found in large quantities in many febrile diseases. Acetone, diacetic, and  $\beta$ -oxybutyric acids are products formed in the incomplete metabolism of fat. Normal combustion of fat can not take place unless there is at the same time combustion of carbohydrates. The substances mentioned are therefore found in the urine whenever an insufficient amount of carbohydrate is ingested, or when the amount ingested can not be utilised. In acute diseases these substances are present for the first reason mentioned; in diabetes, for the second reason. There is no connection between acetonuria and the nervous symptoms accompanying fever.

Acetone, diacetic, and  $\beta$ -oxybutyric acids are regularly found in the urine of patients suffering from cyclic vomiting; they are probably a result, not the cause, of the attacks. In progressing cases of diabetes and in diabetic coma these substances are present in large amount.

#### ANURIA.

By this term is meant an arrest of the urinary secretion. To that form which occurs in the course of renal disease the term "suppression" is generally applied. Anuria is to be carefully distinguished from retention, from the scanty secretion which occurs whenever food is refused or withheld on account of illness, and also from that which accompanies acute diarrhoea, with large, watery discharges. Anuria is sometimes seen in the newly born, where it depends upon some malformation of the genital tract; or, more frequently, upon uric-acid infarctions in the kidneys. The first urine passed after such an attack is very often highly acid, and may contain an abundance of uric-acid crystals and larger masses visible to the naked eye. Other cases admit of no such explanation. For the time, the secretion appears to be completely arrested, as the bladder, both by palpation and catheterisation, is found to be empty. This condition is uncommon in infancy, but it may continue for from twelve to thirty-six hours. So long as infants appear to be perfectly normal in every other respect, the suspension

of the urinary secretion even for twenty-four hours need excite no anxiety.

The treatment consists in the administration of sweet spirits of nitre, in combination with the acetate or citrate of potash, and plenty of water. To a newly-born infant one minim of the nitre and one grain of the citrate of potash may be given every hour or two, in water, until the urinary secretion is established, which will usually be in six or eight hours. If the urine is very highly acid, and stains the napkins, the potash should be continued for several days. Hot fomentations over the kidneys may be used.

#### DIABETES INSIPIDUS (POLYURIA).

This is a chronic disease characterised by the excretion of a very large amount of pale urine of low specific gravity. It is invariably accompanied by polydipsia. The disease is an exceedingly rare one in children.

The exact pathology of diabetes insipidus is not known; but from the conditions under which it occurs it is believed to be a neurosis.

**Etiology.**—Of eighty-five cases collected by Strauss, twenty-one were in children under ten years of age and nine under five years. In Roberts's collection of seventy cases, the disease began in twenty-two children before ten years, and in seven during infancy. In some cases it begins soon after birth. Males are more frequently affected than females, and in certain cases heredity is an important factor. Weil has published a remarkable example of the disease existing in many members of a single family. Falls or blows upon the head, concussion of the brain, tumours of the brain, especially of the occipital region, or chronic hydrocephalus, all have been found associated with diabetes insipidus. It sometimes has followed the acute infectious diseases; but in many cases no cause whatever can be found.

**Symptoms.**—The quantity of urine is enormous, usually exceeding even that in diabetes mellitus. From five to twenty pints daily may be passed. The urine is pale, the specific gravity from 1.001 to 1.006, and it contains neither albumin nor glucose. In a few cases the presence of inosite (muscle sugar) has been found. Restricting the amount of fluid taken causes a very marked diminution in the amount of urine. The intense thirst leads patients to drink enormously of water and other fluids.

Nervous symptoms are usually present. There may be disturbed sleep from the frequent micturition, palpitation, flushing of the face and other vaso-motor disturbances, headache, restlessness, and neuralgia. There may be incontinence of urine. The bladder sometimes becomes

enormously distended. In one of my cases it held forty-five ounces and reached above the umbilicus. The skin is pale and dry, and perspiration is scanty. The general health may not be disturbed. In most cases, however, it is somewhat affected, and there may be the usual symptoms of malnutrition, and even neurasthenia. If it affects young children, their growth may be retarded. The appetite usually remains quite good. The temperature is at times slightly subnormal. The course of the disease is indefinite. It is very chronic, and may last for many years, death taking place only from intercurrent affections.

**Prognosis.**—A few of the cases recover spontaneously. Those of short duration are often cured by treatment. Of the chronic cases in which the disease is well established very few are controlled. The prognosis is worse if there are marked disturbances of the digestive tract or organic brain disease.

**Diagnosis.**—This is easily made from the two marked symptoms, excessive thirst and polyuria. From diabetes mellitus it is easily distinguished by the lower specific gravity and the absence of sugar from the urine. In older children, chronic nephritis with contracted kidney may be confounded with it.

**Treatment.**—Fluids should be moderately restricted. It is a serious mistake to reduce the quantity of fluids too much, since the drinking is not the cause of the diuresis. The diet should be simple and nutritious. The general treatment should be directed to the condition of malnutrition. The clothing should be warm, and a moderate amount of exercise should be allowed. Drugs, in most cases, are of little use; but decided improvement has sometimes followed the prolonged use of codeine; other cases have been benefited by the bromides and belladonna. Treatment must be continued for many months to be of any value.

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## CHAPTER II.

### *DISEASES OF THE KIDNEYS.*

#### MALFORMATIONS AND MALPOSITIONS.

MALFORMATIONS of the kidney are not infrequent. In seven hundred and twenty-six consecutive autopsies at the New York Infant Asylum malformations of the kidney or ureters were met with in seventeen cases. This does not represent the actual frequency with which they occur, for in about half that number of autopsies in two other institutions only a single example was seen. Adding to the cases mentioned two others seen elsewhere, there are twenty cases of renal malformation of which I have notes, classed as follows:

|  |          |
|--|----------|
| Fusion of the kidneys, or horseshoe kidney . . . . . | 4 cases. |
| Supernumerary ureters . . . . .                      | 4 "      |
| Hydronephrosis (alone) . . . . .                     | 8 "      |
| Congenital cystic kidney (alone) . . . . .           | 2 "      |
| Hydronephrosis and cystic kidney . . . . .           | 1 case.  |
| Single kidney . . . . .                              | 1 "      |

In all malformations the left kidney is much more frequently affected than the right, the proportion being nearly two to one. Malformations are more often seen in males than in females. Only two of these conditions are of clinical importance—viz., cystic degeneration and hydronephrosis.

**Cystic Kidneys.**—Two varieties of this malformation are met with. In one the cysts are few in number and large; in the other they are very numerous and small. When the cysts are large the renal tumour may fill the abdominal cavity, even interfering with the birth of the child. The condition is generally bilateral, and the patients die in early infancy. The more common form, that with small cysts, also affects both sides as a rule. The organ often is not enlarged, and it may even be smaller than normal. The surface of the kidney is studded with small cysts, which usually vary in size from a pin's head to that of a pea. The entire organ may consist of nothing but a mass of cysts, held together by loose connective tissue. In other cases the cysts are less numerous, and much renal tissue remains. The cysts are formed by the dilatation of the uriniferous tubules owing to occlusion, which occurs in the development of the kidney. The large cysts are recognised as abdominal tumours; the small ones usually give no symptoms, and are found accidentally at autopsy in patients dying from other diseases.

**Hydronephrosis.**—Of the thirteen cases of which I have notes, this existed as the principal deformity in eleven. In two cases it was associated respectively with cystic degeneration of the opposite kidney and horseshoe kidney. In seven cases only the left side was affected; in six there was double hydronephrosis. Nine patients were males and four females. Seven died before they were six months old, and only two lived to be two years old. This condition is undoubtedly the result of some obstruction to the outflow of urine in the ureter, bladder, urethra, or prepuce, but in only three of my cases could there be found an obstruction sufficient to explain the deformity. In five there was marked hypertrophy of the bladder. In no case was a calculus found as the cause of the obstruction. In most of the cases the ureter was dilated to a diameter of from one-fourth to one-half inch, and in five it was so large as to be easily mistaken for the intestine. Usually the ureters appear much elongated and sacculated; the pelvis and the calices of the kidney may be slightly dilated or the greater part of the kidney may be destroyed, leaving only a series of communicating pockets surrounded by a thin



cortex of renal tissue. After a time chronic nephritis usually develops. This may involve both kidneys, even though the hydronephrosis is uni-



FIG. 102.—CONGENITAL HYDRONEPHROSIS, DILATED URETERS, AND HYPERTROPHIED BLADDER. (From a child one month old.)

lateral. In two cases, typical examples of the atrophic form (contracted kidney) were seen, one of these children dying at the age of one month.<sup>1</sup> The organs are shown in Fig. 102.

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<sup>1</sup> This was in every way a remarkable case. The child died apparently of marasmus. There was double hydronephrosis, the ureters being three-fourths of an inch in diameter. The right kidney was nodular upon the surface, and had a very adherent capsule. Just beneath the capsule there were small cysts containing pus. The left kidney was the seat of hydronephrosis, only its cortex remaining, this being about one-sixth of an inch in thickness. Microscopical examination showed great thickening of the capsule of the right kidney, and several small abscesses situated in the cortex just beneath the capsule. The rest of the kidney was converted into a mass of dense fibrous tissue in which were scattered many uriniferous tubules. The

Urinary symptoms are noted in but few cases during life, and the diagnosis is seldom made. The cause of death is usually some inter-current disease.

Double hydronephrosis is generally associated with, or results in, such changes in the kidneys that the patients die during infancy. It may give rise to one or more tumours, which sometimes attain a large size. Even when renal tumours can not be made out, the hypertrophied bladder may be felt as a hard globular tumour in the hypogastrium. Changes in the urine may not be present until the disease is very far advanced. There may be the general and local symptoms of chronic diffuse nephritis, or, when infection of the genital tract occurs, there are added the symptoms of pyelitis. In the great majority of cases the condition is unrecognised, the patient dying of some disease not perhaps in itself fatal, but rendered so by the condition of the kidneys.

If hydronephrosis is unilateral there may be no symptoms until the dilatation of the pelvis of the kidney has reached a sufficient size to form an abdominal tumour. In most of the cases in children this condition has been noted between the third and the eleventh years. This tumour may be situated in the lumbar region, or it may fill the abdomen. It is cystic, and may be confounded with a dermoid cyst of the ovary. On aspiration a fluid is withdrawn which may be clear, or of a brownish colour, and recognised as urine by the fact that it contains urates and urea. After aspiration the urine passed *per urethram* may be bloody. Aspiration affords only temporary relief, as the tumour quickly refills. If an incision is made and the kidney drained, a cure may result with the formation of a fistula. This may continue indefinitely, or infection of the fistulous tract may occur and suppurative nephritis be set up, which speedily carries off the patient. A better operation is nephrectomy, which may result in a permanent cure if the opposite kidney is healthy, which is usually the case if the child is over three years of age, for the reason above stated, viz., that a child with malformation of both kidneys usually dies in infancy.

**Movable Kidney.**—This is a rare condition in young children. Comby has collected eighteen cases, of which sixteen were in girls and two in boys. Movable kidney was recognised before the tenth year in eight cases, and in two of these before the fourth month. It has been ascribed to too long a pedicle, which may be congenital; also to pressure from abdominal tumours, and to injury. The most important symptoms are

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left kidney was the seat of chronic diffuse nephritis of the atrophic variety, with well-marked changes in the medullary portions. The cortex showed much exudation and less atrophy, being nearly normal in thickness. The small size of the organ was due chiefly to atrophy of the pyramids. The walls of the bladder were greatly hypertrophied, being in places one-fourth of an inch thick. The urethra and prepuce were normal.

paroxysmal pain, which may follow exertion, and a movable tumour. A twist in the ureter may produce hydronephrosis.

#### URIC-ACID INFARCTIONS.

These consist in a deposit in the straight tubes of the kidneys of uric acid or of amorphous or crystalline urates; usually both kidneys are affected, and all the pyramids of each kidney. The infarctions appear to the naked eye as fine, brownish-yellow, fan-shaped striae. Associated with them there may be granular deposits of uric-acid salts in the pelvis of the kidney, and sometimes evidences of catarrhal inflammation of the pelvis, including even the presence of blood. This condition probably occurs, to some degree, at least, in nearly all infants during the first ten days of life. It was formerly supposed that the discovery of these appearances was proof that an infant had breathed, and a certain medico-legal importance was therefore attached to them. This is now known not to be the case, as they are sometimes found in still-born infants.

The cause of this condition is the excretion of uric acid before there is sufficient water to dissolve it, so that the crystals are deposited in the tubes. Uric-acid infarctions are found chiefly in children dying before the end of the second week, although it is not uncommon to see them as late as the third or fourth or even the sixth month. In most of the cases, as the urinary secretion becomes more abundant, the deposits are washed out in the urine and appear as brownish-red or pink stains upon the napkins. Infarctions may give rise to a slight inflammation of the renal tubules, but very rarely to any serious lesion; sometimes they remain as deposits in the calices or the pelvis of the kidney or in the bladder, forming the nucleus of a calculus. The symptoms to which they give rise are mainly scanty urination during the first week of life, and occasionally anuria for the first day or two. Sometimes there is evidence of severe pain; priapism may be present, and there is the stain upon the napkin already referred to. The treatment is to give water freely and some alkaline diuretic such as citrate of potash. One grain should be given every two hours until the secretion is fully established; this in most cases will be within twenty-four hours.

#### CHRONIC CONGESTION OF THE KIDNEY.

This results from interference with the return circulation of the kidney, and may be caused by congenital malformation or valvular disease of the heart, chronic broncho-pneumonia or chronic pleurisy; also by the pressure of any abdominal tumour upon the inferior vena cava or the renal veins.

The kidneys are generally enlarged, firmer than normal, and dark-coloured. All the capillary vessels are swollen and distended with blood, and their walls are thickened. In addition to the symptoms of the pri-

mary disease, the amount of urine passed is usually scanty and of high specific gravity. Albumin and casts are generally present, but are not constant. The treatment should be directed toward the primary condition, and, in addition, an effort should be made to increase the urine by alkaline diuretics, caffeine, digitalis, and diuretin.

#### ACUTE DEGENERATION OF THE KIDNEYS.

In the succeeding pages devoted to the kidney I have followed in the main Prudden's classification.

In acute degeneration of the kidney the principal or only change is in the epithelium of the tubules. It is exceedingly common both in infancy and in childhood, being found to a greater or less degree in all autopsies upon patients dying of acute infectious diseases, but it is most marked in cases of scarlet fever, diphtheria, and acute pleuro-pneumonia. It may be found in any disease characterised by prolonged high temperature; and it is the explanation of the cases of so-called febrile albuminuria. The cause is in all probability direct irritation of the epithelium of the tubules by the toxins eliminated by the kidneys. It may also be induced by irritating drugs, such as cantharides or turpentine. By some writers these cases have been classed as examples of acute nephritis; hence the great discrepancy which exists in statements made as to the frequency of nephritis in the different infectious diseases.

The kidneys are usually slightly enlarged, softer, and paler than normal. On section the cortex may be somewhat thickened, and the straight tubules marked by yellowish-gray lines. It is the appearance commonly spoken of as cloudy swelling. The kidneys are seldom much congested. The microscope shows a granular degeneration and death of the epithelium of the tubules, and when severe this may be accompanied by congestion and the exudation of serum.

Acute degeneration of the kidneys gives rise to no symptoms in addition to those of the original disease, except the appearance of a moderate amount of albumin in the urine, with a few hyaline, granular, or epithelial casts. It can not be said that such a condition adds much to the danger from the original disease. In cases that recover, the condition of the kidney becomes entirely normal. The development of the symptoms of degeneration of the kidneys in infectious diseases calls for no special treatment beyond a continuance of the fluid diet.

#### ACUTE DIFFUSE NEPHRITIS.

(*Acute Interstitial Nephritis; Acute Exudative Nephritis; Glomerulo-nephritis; Acute Bright's Disease.*)

**Etiology.**—This variety of nephritis occurs apparently as a primary disease both in infants and in older children. Most such cases are un-

doubtedly of infectious origin, although the point of entrance of the infection may be difficult or impossible to determine. Acute diffuse nephritis is very frequently secondary to the acute infectious diseases, especially to scarlet fever and diphtheria. It occasionally follows measles, varicella, empyema, typhoid fever, acute diarrhoeal diseases, pneumonia, meningitis, influenza, and malaria. It is the characteristic variety of secondary nephritis occurring in severe septic conditions. The exciting cause of the inflammation is in some cases the irritation from toxins; but usually there is in addition the entrance of pathogenic organisms carried by the circulation. Thus in post-scarlatinal nephritis, of which the one under consideration is the characteristic form, the cause is now generally admitted to be the toxins of the primary disease, to which in many cases is added infection by the streptococcus. While nephritis is more frequent after severe attacks of scarlet fever, it may occur after those which are very mild, even when patients have been kept in bed throughout the disease. The frequency of nephritis as a sequel of scarlet fever varies much in different epidemics; the average is from six to ten per cent. I have seen two cases of acute nephritis in infants, the apparent cause of which was the irritation of a highly concentrated urine. This was the result of the infants taking for a long time very little food, and almost no water.

**Lesions.**—In severe cases the kidneys are usually enlarged, soft, and oedematous. The capsule is non-adherent. The cortex is thickened, either reddened or pale; frequently it is mottled with red, owing to the presence of small hæmorrhages. There may be congestion of the entire organ; or the pyramids may seem unusually red by contrast with the pale and thickened cortex.

All the structures of the kidney—glomeruli, tubular epithelium, and interstitial tissue—are involved in the inflammatory process. The cells covering the glomerular tufts of capillaries are swollen and proliferated. They have frequently undergone fatty degeneration and separated. The epithelial cells lining Bowman's capsule may undergo the same changes, but usually to a lesser degree. The space between the capsule and the tuft may contain exfoliated epithelium in considerable quantity, also cell-detritus, albuminous (granular) exudate, leucocytes, and red blood-cells. The tubular epithelium undergoes albuminous and fatty degeneration and may desquamate. Thus the tubules may contain epithelial fragments, serum, red blood-cells, leucocytes, and casts. The interstitial connective tissue is infiltrated with serum and in places with small round cells. In cases of longer duration a general increase of the connective tissue may take place, which is permanent.

When the glomerular changes are especially marked, as in acute nephritis following scarlet fever, the process is often spoken of as *glomerulo-nephritis*. If the degeneration of the tubular epithelium is

extreme, as in severe cases of diphtheria dying shortly after the onset, the nephritis may be described as the *parenchymatous* or *degenerative* type. In the *hemorrhagic* form there are hæmorrhages into the tubules, glomeruli, or interstitial tissue. In infants and young children the *exudative* type of acute diffuse nephritis is especially frequent. In this there is an exudative inflammation with large accumulations of leucocytes, serum, and red blood-cells in the glomeruli and tubules, the parenchyma and interstitial tissue sometimes being markedly and sometimes but slightly changed. Should the interstitial tissue suffer early and severely, the nephritis becomes of the *productive* or *interstitial* type. This form is most frequently seen with severe, protracted cases of scarlet fever and diphtheria,<sup>1</sup> especially in older children. It sometimes occurs as an apparently independent process.

**Symptoms.**—1. *Primary Form in Infants.*—These cases are not common, and the symptoms are so obscure that they are often overlooked. At least ten such cases have come under my observation. The inflammation in most of them was of the exudative type.

The onset in nearly every instance was abrupt, usually with high fever and vomiting, the temperature being in several cases over 104° F. Dropsy was very exceptional, being noted in but six cases; in most of these it was slight, and seen only toward the close of the disease. Fever was present in all cases. In those observed by myself it was high and irregular in type, ranging from 101° to 105° F. The duration of the disease was from eight days to four weeks, the average being about two and a half weeks. Vomiting and diarrhœa were noted in half the cases, but were rarely prominent, and marked either the onset of the attack, or were traceable to indigestion accompanying the fever; very rarely did they exist as symptoms of uræmia. Anæmia was a prominent symptom in nearly every case, and it was this which enabled me in several instances to make a correct diagnosis. Nervous symptoms were usually prominent. In several patients there was dyspnoea without pulmonary disease, partly due, no doubt, to the anæmia. In nearly all cases there was marked restlessness or muscular twitchings, and in three there were convulsions. Dulness and apathy were present in the majority of the fatal cases, but deep coma was never seen. Several patients presented the typical symptoms of the typhoid condition. The urine was rarely scanty until near the close of the disease, and sometimes not even then. Suppression of urine occurred in but a few cases. Albumin was frequently absent early in the attack, but was invariably present at a late period, although rarely in large amount. Casts were found in all cases that were carefully examined microscopically. They were not usually numerous,

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<sup>1</sup> Councilman, Mallory, and Pearce, *Diphtheria: A Study of the Bacteriology and Pathology of Two Hundred and Twenty Fatal Cases*, 1901.

and were chiefly of the hyaline, granular, and epithelial varieties. No blood casts were seen. There were usually many pus cells and renal epithelial cells, together with red blood-cells in moderate numbers.

Of the twenty-four cases, sixteen died and eight recovered. Of my own ten cases, nine were fatal, the diagnosis being confirmed by autopsy in every case but two. Whether these figures represent the actual mortality of the disease it is difficult to say. No doubt there are many mild cases which are unrecognised. The severe ones, however, are quite uniformly fatal, chiefly on account of the tender age of the patients.

2. *Primary Form in Older Children.*—This also is a rare form of renal disease. As compared with the same condition in infants, the onset is usually less abrupt, the febrile symptoms are less marked, and the termination is less frequently fatal. Dropsy is rarely marked, and often there is none at all. The urine is only slightly diminished in quantity; the amount of albumin is small; casts are not numerous, and usually hyaline, epithelial, or granular; very rarely is there much blood present. Uræmia is infrequent, and the prognosis is better than in infancy.

The interstitial type may begin abruptly with febrile symptoms, dropsy, headache, lumbar pains, scanty urine, and often with vomiting; or it may come on somewhat insidiously with few constitutional symptoms, but with dropsy and changes in the urine.

3. *Secondary Form.*—The secondary nephritis of acute infectious diseases may occur at the height of the febrile process or at a later period, and its severity is generally proportionate to the intensity of the infection. The general symptoms of nephritis are often not marked, and dropsy is rare; so that unless the urine is examined the condition may be overlooked. The urinary changes are essentially the same as those already mentioned in the primary cases. Suppression of urine and the development of the symptoms of acute uræmia are infrequent. While nephritis adds considerably to the danger from the primary disease, it is seldom itself the cause of death, although this is sometimes the case in scarlet fever or diphtheria.

The characteristic type of nephritis which follows scarlet fever most frequently develops during the third or fourth week of the disease. The onset may be gradual, dropsy being first noticed. Or it may begin abruptly without dropsy, but with headache, vomiting, scanty urine, fever, and even convulsions. The temperature generally ranges from 100° to 101.5° F., but in very severe attacks it may be 104° or 105° F. While dropsy is usually present, it may be slight or absent in severe and even in fatal cases. It is first seen in the face, next in the feet, legs, and scrotum; there may be general anasarca, with dropsy of the serous cavities of the body, the pleura, or the peritonæum, rarely the pericardium. As the disease progresses there is always a very marked degree of anæmia.

The urine is, as a rule, greatly diminished in quantity, and may be

suppressed. Albumin is invariably present, although not always at first; it is usually in large amount, often enough to render the urine solid upon boiling. The urine is of a dark, reddish-brown or smoky colour, owing to the presence of red blood-cells or hæmoglobin. The total amount of urea eliminated is far below the normal. The specific gravity may be low, even though the quantity is very small. Casts are present in great numbers, chiefly hyaline, granular, and epithelial casts from the straight tubes; not infrequently there are blood casts. Red blood-cells are present in great numbers; also many leucocytes, and renal epithelium.

The duration of the active symptoms in cases terminating in recovery is from one to three weeks. The temperature and dropsy gradually subside. Improvement in the urine is shown by an increase in quantity, by an increased elimination of urea, and by a diminution in the amount of blood, albumin, and the number of casts. A few casts may persist for several weeks, and a small amount of albumin for two or three months.

In the graver cases, when the onset is accompanied by high temperature, pain in the back and loins, and a rapid, full pulse of high tension, the urine is very scanty and is often suppressed. Then follow the symptoms of uræmia. In children this is usually manifested by vomiting, great restlessness or apathy, and often by diarrhœa. Less frequently there is headache, dimness of vision, stupor developing into coma, or convulsions. If the secretion of urine is re-established, the nervous symptoms abate and the patient may recover. This has been known to occur after complete suppression has lasted thirty-six hours. Care should be taken not to mistake retention for suppression. If doubt exists, percussion of the bladder and the use of the catheter will quickly settle the question.

There are several complications for which the physician must constantly be on the lookout during attacks of acute nephritis; the most frequent are pneumonia, pleurisy, pericarditis, and endocarditis; more rarely there may be meningitis and œdema of the glottis. It is from complications or acute uræmia that death usually occurs.

**Prognosis.**—This is to be considered from two points of view: first, the danger to life during the acute stage of the disease, and, secondly, the danger of the development of chronic nephritis. The great majority of patients survive the acute stage, and not infrequently even those recover who have presented grave symptoms of uræmic poisoning. The quantity and specific gravity of the urine, and the number and variety of the casts, are a much better guide in prognosis than the amount of albumin. The existence of severe nervous symptoms, such as stupor, intense headache, dimness of vision, and persistent vomiting, add much to the gravity of the case, as does also the presence of any serious complication. In general it may be said that if there is no suppression of urine, or if there are no symptoms of uræmia and no complications, recovery is



almost certain if the child is over three years old; in younger children the outlook is less favourable. The general opinion prevails that acute diffuse nephritis in childhood, whether it is primary or occurs as a complication of scarlet fever, is rarely followed by the chronic form of the disease; and such was the view I formerly held. Larger experience, however, has convinced me that this sequel is not very uncommon. The interval of apparent health may sometimes cover a period of several years, and the later nephritis may be attributed to other causes; but all cases of scarlatinal nephritis should be carefully watched for a long time, and after a severe attack a guarded prognosis should always be given as regards the ultimate result.<sup>1</sup>

**Treatment.**—Prophylaxis is important, and relates principally to the secondary form which occurs in the course of infectious diseases, especially post-scarlatinal nephritis; but the measures here outlined apply equally to all varieties. The inflammation of the kidney being in most of these cases the result of direct irritation by the toxins which are eliminated by them, it follows that elimination through the skin and intestines should be increased, and that the urine should be rendered as little irritating as possible by largely increasing its quantity. The first indication is met by frequent sponging, warm baths, and keeping the bowels freely opened by saline cathartics, sufficient being given to produce one or two loose movements daily. To meet the second indication, the patient should be kept upon a diet of milk and farinaceous food, at least for the three weeks of the disease, and, if possible, for a full month. At the same time he should drink very freely of alkaline mineral waters, or of plain water. If milk is not well borne, kumyss, whey, buttermilk, or junket may be used, or thin gruels mixed with milk. When the first trace of albumin appears in the urine this plan of treatment should invariably be followed. In addition to these measures, after an attack of scarlet fever the patient should be kept in bed for at least a week after the temperature has become normal.

The mild cases of acute nephritis tend to spontaneous recovery under the hygienic and dietetic treatment outlined, i. e., rest in bed, the diet mentioned, the drinking of large quantities of water, and attention to the action of the skin and bowels. These measures should be continued so long as the urine contains any considerable amount of albumin, or so long as the patient's general condition will permit. Should he become

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<sup>1</sup> The following case may be cited as an illustration of this point: A girl at the age of seven years had scarlet fever, followed by nephritis; the dropsy having lasted, it was reported, for three months. She was believed to have recovered perfectly, and remained in apparent health until she was sixteen, when, as a supposed result of a severe chilling, she developed dropsy and all the symptoms of acute nephritis. From that time, although she lived for three years, and was often for months at a time seemingly in the best of health, her urine was never free from casts and albumin, and she finally died in uræmic convulsions.

very anæmic, or lose much in weight, it may be necessary to enlarge the diet by the addition of more solid food. An increase in the diet and exercise should be made very gradually, and the effect upon the urine carefully watched.

The severe cases, with scanty urine, fever and marked dropsy, require more active treatment. Free diaphoresis should be maintained by the hot pack or vapour bath. Active counter-irritation should be maintained over the kidneys by dry cups followed by poultices, or the mustard paste. Two or three loose movements from the bowels should be secured by the administration of calomel or, better by Rochelle or Epsom salts. Harm is sometimes done by carrying this depletion too far, and its effect upon the patient's general condition must be closely watched. If suppression of urine occurs with the development of uræmic symptoms—delirium, high temperature, flushed face, vomiting, and a pulse of high tension—nitroglycerin is indicated; a child of five years may take gr.  $\frac{1}{10}$  every hour for five or six doses, or until an effect is produced.

In addition to these measures rectal injections of a normal salt solution should be given high in the colon, at a temperature of from 104° to 108° F. At least two quarts should be given several times a day, to be continued until a free flow of urine is established. This is one of the most valuable means we possess of increasing elimination by the kidneys and skin.

The nervous symptoms of uræmia are best relieved by chloral, which should be given per rectum. When such symptoms are marked, from six to ten grains are required for a child of five years, to be repeated in two hours if no improvement is seen. Uræmic convulsions may sometimes be averted by the use of morphine hypodermically. In extreme conditions not relieved by the measures mentioned, venesection should by all means be practised; from three to six ounces of blood may be drawn from a child of five years, according to his general condition and the urgency of the symptoms. The depressing effect may largely be overcome by immediately following this with an intravenous injection of a normal salt solution. Twice as much as the fluid drawn should be introduced. This will almost invariably give at least temporary relief, which may afford time for the operation of other measures such as catharsis and diaphoresis. Pulmonary œdema is no contra-indication to bleeding; the best of all guides as to its use is a pulse of very high tension.

One should always be on the lookout for complications, especially dropsy of the serous cavities, pericarditis, and œdema of the lungs. Convalescence is nearly always slow, and a patient who has suffered from nephritis needs careful attention for a long time. Anæmia is always present, and iron is required. The diet should be carefully restricted for several months; much nitrogenous food should be avoided. If the disease tends to pass into a subacute form, the child should, if possible,

be sent to a warm climate, and kept there during the succeeding winter, and every means taken to improve the general nutrition. Flannels should be worn next to the skin, and special precautions taken against any exposure which might cause an exacerbation of the disease.

#### CHRONIC NEPHRITIS.

Chronic inflammation of the kidney is an infrequent condition in childhood. In infancy it is almost unknown, except in connection with congenital hydronephrosis or other malformations of the kidney. Two pathological varieties are met with: (1) chronic diffuse nephritis of the parenchymatous or degenerative type; (2) chronic diffuse nephritis of the interstitial or productive type. As the disease progresses the former may assume the characteristics of the latter variety.

**Etiology.**—Chronic nephritis is most frequently seen as a sequel of the acute nephritis of scarlet fever, less often after other acute infections. The only other important causes in early life are hereditary syphilis, chronic tuberculosis, and valvular disease of the heart. Nearly all the cases occur in children over five years of age.

**Lesions.**—The lesions of chronic nephritis in childhood do not differ essentially from those seen in later life. In the chronic parenchymatous type the kidneys are usually enlarged, the surface is smooth or slightly nodular, and the thickened cortex yellowish-white on section. These are often called "large white kidneys." On the other hand, the kidneys may be nearly normal in appearance, or smaller and with a thinner cortex than is usual. In the so-called "large red kidneys" the cortex is red or mottled red and yellow, owing to hæmorrhages into the tubules or interstitial tissue. The microscope shows that the renal epithelium is swollen, granular, fatty, and degenerated. The tubes contain leucocytes, red cells, cast matter, and the detritus of broken-down epithelial cells. In some places they are dilated, in others atrophied. In the glomeruli there is a growth of capsule cells, compression and atrophy of the tufts, with the formation of new connective tissue.

In the chronic diffuse nephritis of the interstitial type (granular kidney) the organs are smaller than normal, with a nodular surface and adherent capsule. The cortex is thinned, and the colour is gray or red. In addition to the lesions found in the preceding variety, there is an extensive production of new connective tissue, which is irregularly distributed throughout the kidneys. The tubules in some places are dilated to form cysts of considerable size, while in others they have completely disappeared. The glomeruli may be atrophied to little fibrous balls; or if chronic congestion has preceded the inflammation, some may be large and the capillaries dilated with hyaline degeneration of their walls.

**Symptoms.**—1. *Chronic Nephritis of the Parenchymatous Type.*—

This form of the disease may be chronic from the outset, or follow an acute attack from which the patient is often believed to have recovered completely. The symptoms sometimes immediately follow the acute attack; at others there is an interval of apparent recovery, extending over a few months or even years. Very rarely no such history of an antecedent acute attack can be obtained, and the symptoms come on gradually and insidiously. Such cases occur chiefly in older children, and their clinical features do not differ essentially from those of adult life.

As a rule dropsy is present, although it is variable in amount, and fluctuates considerably from time to time. There may be not only œdema of the cellular tissue, but effusion into the pleura, peritonæum, and even the pericardium. As the case progresses, anæmia is always a marked symptom. There are various disturbances of digestion—loss of appetite, occasional vomiting, and attacks of diarrhœa. From time to time nervous symptoms may be quite prominent, such as headaches, sleeplessness, neuralgia, fatigue upon slight exertion, and dyspnoea. Attacks of epistaxis are not infrequent.

For the greater part of the time the urine contains albumin and casts. They vary much in amount at different periods in the disease, according to the rapidity of its progress. During periods of exacerbation, both albumin and casts are very abundant, while in the intervals the amount of albumin may be small and the casts few. The casts are hyaline, granular, epithelial, and fatty. The daily quantity of urine is much reduced during the periods of exacerbation, while at other times it may be nearly normal. The specific gravity is usually normal or high.

If amyloid degeneration is present, there are generally associated with the renal symptoms, others dependent upon amyloid changes in other organs. The spleen and liver are enlarged; there may be ascites and diarrhœa, and there is usually present a peculiar alabaster cachexia.

The duration of this form of chronic nephritis depends much upon the surroundings of the patient and the treatment. It is rarely shorter than two years, and it may last for many years. The progress is always irregular, and marked by periods of exacerbation and remission. The patients die from acute uræmia, from some intercurrent disease, or from complicating pneumonia, pleurisy, pericarditis, endocarditis, or from pulmonary œdema.

2. *Chronic Nephritis of the Interstitial Type.*—This is a very rare disease in early life, being much less frequent even than the preceding variety of nephritis. In some cases there is a history of hereditary syphilis; in others, of chronic alcoholism. The early symptoms are few, and the disease usually develops insidiously. The urine is pale, excessive in amount, and of low specific gravity—1.001 to 1.008. Albumin

is often absent, and, when found, the quantity is small. Dropsy likewise is rare, and never marked. Nervous symptoms are often prominent, such as headache, attacks of spasmodic dyspnoea resembling asthma, neuralgias, and disturbances of vision. High arterial tension and hypertrophy of the left ventricle are regular symptoms; and even atheromatous degeneration of the arteries may be present. Dickinson reports an instance of this in a patient only six years of age. Late in the disease, hæmorrhages may occur, and these may be the cause of death. Filatoff has reported a cerebral hæmorrhage in a child of eleven years. Acute uræmia is, however, the usual termination of this form of nephritis. The course is slow, and the disease may be overlooked until the final uræmic symptoms occur.

**Prognosis.**—The prognosis of chronic nephritis as to complete recovery is always unfavourable; and although cases are seen in which symptoms are absent for several years, they almost invariably return. As to the duration of the disease, no exact prognosis can be given, because from the symptoms, it is difficult or impossible to determine exactly the extent of the disease in the kidney and the rapidity of its progress. The continued passage of a large amount of urine of low specific gravity is invariably to be interpreted as evidence of fibroid changes in the Malpighian tufts, and is a bad symptom. A large amount of dropsy, the coexistence of valvular disease of the heart, and marked renal insufficiency, as shown by the quantitative examination of the urine, are all very unfavourable symptoms.

**Diagnosis.**—Chronic nephritis, like the acute forms, is likely to be overlooked because of the failure to examine the urine in children. Regular and frequent examinations should be made in all cases of convulsions, of persistent or frequent headaches, severe anæmia, hypertrophy of the heart, high arterial tension and of general malnutrition, as well as when the more obvious symptoms of renal disease, such as dropsy and scanty urine, are present. Nor should one be too ready to make the diagnosis of functional albuminuria because he finds albumin only occasionally and in small quantity. All such cases demand most careful observation and the closest attention for a long period before excluding organic renal disease.

**Treatment.**—Children with chronic nephritis are to be treated on the same general plan as adults. The purpose of treatment is to retard as much as possible the progress of the disease and to relieve the symptoms as they arise. It is of the greatest importance to remove the patient from conditions in which exacerbations are liable to occur. If it is possible, he should be sent to a warm, dry climate in winter, and all exposure to cold avoided; an out-door life is desirable. Most patients require general tonic treatment with very moderate but regular exercise, never carried to the point of fatigue, as much rest as possible in a recumbent

position, a fluid diet, consisting largely of milk as long as this can be borne, and the administration of iron. Dropsy calls for a salt-free diet, diuretics, saline cathartics, and vascular stimulants. If uræmia develops, with high arterial tension and stupor, headache, and convulsions, venesection should be resorted to, or nitroglycerin used. Morphine may be given hypodermically if the nervous symptoms are very marked.

Decapsulation of the kidney is to be considered in cases growing progressively worse in spite of medical treatment. The immediate risks of the operation are rather less than would be expected. I have seen striking temporary benefit in several cases when this operation was done upon young children. In no case, however, was the improvement permanent, all the patients dying within a year after it was performed.

#### TUBERCULOSIS OF THE KIDNEY.

In general tuberculosis, miliary tubercles are frequently seen both upon the surface of the kidney and in its substance. These give rise to no symptoms and are of no clinical importance. Larger tuberculous deposits are extremely rare in early life. They usually occur in patients who are the subjects of general tuberculosis, and are associated with tuberculosis of other parts of the genito-urinary tract, or they may exist as the primary, or even the only, tuberculous lesion in the body. Ascending infection occurs occasionally but it is rare; nearly all cases are of the descending type, i. e., primary in the kidney. Infection of the kidney therefore generally takes place through the circulation and not from the bladder. Aldibert's figures show that in children the bladder usually escapes even when the kidneys are tuberculous, for of thirteen cases of renal tuberculosis the bladder was involved in but two. The disease when primary begins in the cortex, but soon extends to the mucous membrane of the pelvis and the calices of the kidney, and also to the pyramids. As a rule, but one kidney is affected. The process may be confined to the pyramids, where are found cheesy nodules which may be single or multiple. These ultimately break down and form abscesses. The process may result in almost complete destruction of the pyramids, and even of portions of the cortex, so that the kidney may consist of a mere shell of renal tissue. Suppuration in the neighbourhood of the kidney (perinephritic abscess) often coexists.

The symptoms are quite indefinite. There may be localised pain and tenderness in the region of the kidney, and a tumour if there is perinephritis. The symptoms of irritability of the bladder may be almost as severe as in cases of calculus. Pus usually appears in the urine as a constant symptom, and blood is often present. But the only thing that is diagnostic is the discovery of tubercle bacilli in the urine.

The treatment is the same as in adults.

## MALIGNANT TUMOURS OF THE KIDNEY.

In the great majority of cases tumours of the kidney are malignant. Of fifty-one cases collected by Aldibert which were operated upon, forty-eight were malignant, and three benign.

Malignant growths are almost invariably primary. In children under five years, although not common, they are yet more frequent than any other variety of malignant tumour of the abdomen. The earlier cases reported were classed as carcinoma. It is now well established that carcinoma is very infrequent, and that nearly all the cases are varieties of sarcoma. The tumour grows from the cortex of the kidney, or from the pelvis, sometimes from the adrenals. It may infiltrate the whole kidney, so that there is no trace of renal structure remaining, or it may form an immense tumour on one side of the kidney, which is only partially invaded. These tumours are very rarely cystic, but they are quite soft, and hæmorrhages often occur into their substance. There may be secondary growths in the liver, the lungs, the retroperitoneal glands, in the opposite kidney, in the intestines, or in the pancreas. Pressure of the tumour upon the ureter may lead to hydronephrosis, and upon the inferior vena cava, to thrombosis of that vessel. As it grows, the tumour sometimes becomes adherent to nearly all the abdominal organs by localised peritonitis. It may lead to ascites, but it very rarely causes general peritonitis. The growth may reach a great size, usually from five to fifteen pounds, but in one case reported by Jacobi it weighed thirty-six pounds. In Seibert's collection of forty-eight cases the right kidney was involved in twenty-four, the left in twenty-two, and both kidneys in two cases.

**Etiology.**—These tumours of the kidney may be congenital. This was true of 5 cases in a series of 55 collected by Jacobi. The majority occur in early childhood. In the collection of 130 cases by Longstreet Taylor in which the ages are given, 106 were observed during the first five years, and 57 of these in the first two years of life. The sexes were about equally affected.

**Symptoms.**—The principal symptoms are tumour, hæmaturia, and cachexia. The tumour is usually first noticed. It is in most cases discovered in the loin, but grows forward toward the median line. Its surface may be lobulated and irregular or quite smooth; and although solid, it is sometimes so soft as to give an obscure sensation of fluctuation. It may grow to an enormous size, causing displacement of the liver, spleen, intestines, and lungs. The progress of the growth is usually rapid, so that from the size of a fist, the tumour may grow in the course of five or six months so as to fill the abdomen. By careful palpation it will be found—certainly when the tumour is small—that although it may be quite freely movable, its attachment is near the lumbar spine.

Hæmaturia may be the first symptom noticed. The amount of blood passed is sometimes quite large, but is usually small, and may be discovered only by the microscope. Pain is rare, and is due to localised peritonitis. Constitutional symptoms are absent until the tumour has attained a large size, when a cachexia develops and the patient wastes steadily while the tumour continues to grow. The pressure effects are dyspnoea, from compression of the lungs; œdema of the lower extremities, from pressure upon or thrombosis of the vena cava; vomiting and indigestion, from pressure upon the stomach and intestines. Secondary deposits very rarely cause any symptoms except in the lungs, where they may give rise to cough, and even to hæmoptysis.

The course of the disease is steadily from bad to worse. The usual duration of life in patients not operated upon is from three to ten months after the tumour is large enough to be easily discovered.

**Diagnosis.**—The important points are, the position and attachment of the tumour, its steady growth and solid character, hæmaturia, and the age of the patient (under five years). It may be confounded with hydronephrosis, dermoid cyst of the ovary, enlargement of the spleen, retroperitoneal sarcoma, tumours of the liver, or even of the abdominal wall.

**Treatment.**—Nothing is to be said regarding the medical treatment of these cases. Unless operated upon, they invariably terminate fatally. Some of the results of operation during recent years have been so encouraging that no case should be abandoned, no matter how young the patient, but recurrence in the opposite kidney is probable.



FIG. 103.—SARCOMA OF THE KIDNEY. Child thirteen months old. Weight of tumour, seven pounds. This patient was followed for sixteen years and there was no recurrence.



**Benign Tumours.**—These are distinguished by their slow growth, and by the fact that the constitutional symptoms are mild or wanting. Of the three cases mentioned by Aldibert, one was adenoma, one fibroma, and one was fibro-cystic.

#### PYELITIS—PYELO-CYSTITIS.

Pyelitis is an inflammation of the mucous membrane lining the pelvis of the kidney; cystitis is an inflammation of the mucous membrane of the bladder. They may exist separately or together. With pyelitis there may be inflammation of the ureter or of the kidney itself (pyelonephritis), and it may be acute or chronic. It may result in the accumulation of pus in considerable amount in the pelvis of the kidney (pyonephrosis).

**Etiology.**—The most frequent local cause of pyelitis is irritation from renal calculi. It is also associated with congenital malformations of the kidneys or ureters, with renal tuberculosis and renal tumours. It may result from an extension of inflammation from the tissues surrounding the kidney (perinephritis), or from an abscess opening into the pelvis of the kidney. Acute pyelitis sometimes occurs as a complication of scarlet or typhoid fever, diphtheria, influenza, or pyæmia; but it is also seen apart from these diseases, when it occurs apparently as a primary affection. In most of the severe cases of pyelitis there is also present a certain amount of nephritis.

Acute pyelitis in young children is usually due to an ascending infection from the bladder. In these cases the evidences of inflammation of the bladder are slight or, more frequently, entirely wanting. This form of inflammation occurs almost invariably in female infants. Cultures made from the urine have shown with great uniformity the presence of the colon bacillus. In many of the cases the pyelitis is preceded by an attack of diarrhoea. It is surprising that vulvo-vaginitis is seldom present. It seems quite possible that infection may also occur, especially in male infants, by a direct extension from the intestine to the bladder, or through the blood. Trumpp examined the urine in sixteen patients with gastro-enteritis and found the colon bacillus in thirteen, of whom nine were females.

**Lesions.**—When pyelitis develops from a local cause it is usually unilateral; otherwise both sides are involved. In the cases of acute cystitis or pyelo-cystitis there are the usual appearances of an acute catarrhal inflammation of the mucous membrane, with congestion, swelling, and sometimes minute hæmorrhages. In chronic cases there is thickening and sometimes a granular condition of the lining membrane. There may be an accumulation of pus of considerable size, distending the pelvis and calices (pyonephrosis). If the condition is one depend-

ing upon a calculus or congenital deformity, and in all protracted and severe cases, the kidney itself is involved to a greater or less degree; the extent of the nephritis will depend upon the nature of the exciting cause and the duration of the process.

**Symptoms.**—The history of the following case illustrates the main clinical features of acute infectious pyelitis, in this instance occurring apparently as a primary disease:

A previously healthy female infant of eight months was taken suddenly with a chill, followed by a very high fever. The child was ill for ten days before the nature of the disease was suspected. During this time the temperature ranged between 101° and 106° F., touching 105° nearly every day; but the chill was not repeated. The other constitutional symptoms were not severe. At the first examination of the urine there was found a large amount of pus, which on standing was equal to one-twelfth of the volume of the urine passed; the reaction was strongly acid. There were no signs of vaginitis or vulvitis, no *ardor urinæ*, no evidence of local pain either in the bladder or kidney, no abnormal frequency of micturition, no localised tenderness, and no vomiting. At later examinations there were found in moderate numbers epithelial cells from the bladder, and the tubules and pelvis of the kidney, also a few hyaline casts, but not more albumin than would be explained by the amount of pus. Under no treatment except alkaline diuretics, the temperature gradually fell to normal, and the pus steadily diminished in quantity, and at the end of five weeks had practically disappeared from the urine. A report sixteen months later stated that the child had remained well and entirely free from urinary symptoms.

In some cases there are recurring chills, with wide fluctuations in temperature; in others there may be only pyuria, with moderate fever and few other constitutional symptoms. The course of the temperature is a very irregular one. The fever is seldom continuous, but may be interrupted by periods of normal temperature, lasting several days. The duration of the acute attack may be from two to six weeks, and pus cells may be found microscopically for a much longer time. If the disease complicates one of the acute infectious diseases, pyuria may be the only symptom. If cystitis is also present micturition is frequent, and may be painful. The urine in acute pyelo-cystitis is turbid from the presence of pus, the amount of which may be from one to fifty per cent of the volume of the urine. The amount of pus varies greatly from day to day. It is often abundant when the temperature is low, and almost absent when the temperature is high, this fluctuation depending upon the accumulation or the discharge of the pus. The quantity of urine is generally somewhat diminished, and it may be quite scanty. The reaction is usually acid, even though the amount of pus is large. Albumin is present in proportion to the amount of pus or the degree of nephritis.

Red blood-cells are found under the microscope in most of the very acute cases, and may be in sufficient numbers to colour the urine. The pus cells in recent cases are usually well preserved, but in old cases they may be degenerated. There are many epithelial cells—conical, fusiform, and irregular cells with long tails. There may be renal epithelium and hyaline, granular, or epithelial casts, varying in number with the severity of the nephritis. In a catheterised specimen the colon bacillus is usually present in pure culture.

In chronic pyelitis only pyuria may be present, or there may be a tumour, owing to the pyonephrosis. From time to time, in the chronic form, there may be intermittent attacks of acute pyelitis resembling those above described. In pyelitis depending upon congenital malformations, pyuria is usually the only symptom, unless pyonephrosis is present. With calculi we may have acute or chronic pyelitis; there may be localised pain, tenderness, sometimes a tumour, occasionally hæmaturia, and perhaps a history of renal colic or the passage of gravel. With tuberculosis, there is chronic pyuria and the presence of tubercle bacilli in the urine. The symptoms of general tuberculosis are commonly associated. If there is perinephritis, the inflammation is usually acute, and there are present the local symptoms of the original disease. If an abscess opens into the pelvis of the kidney, there may be a sudden discharge of pus in large quantity with a subsidence of previous local symptoms, including the tumour. With neoplasms, both pus and blood may be found in the urine, but the latter is more frequent.

**Diagnosis.**—The characteristic symptoms of acute pyelitis are chills, which may be repeated, high and fluctuating temperature, scanty urine, frequently pain and tenderness over the kidneys, and pyuria. The diagnosis of pyelitis is made only by an examination of the urine, which should never be omitted in cases of obscure high temperature, even in infancy, particularly if chills are present. When cystitis is associated, the only additional symptoms may be pain and other signs of vesical irritation. These symptoms, with an acid urine containing a large amount of pus and epithelial cells like those described, are sufficient to establish the diagnosis of pyelo-cystitis. If the pus comes from the opening of an abscess into the bladder, ureter, or pelvis of the kidney, the local signs of such abscess will usually be present.

**Prognosis.**—In cases apparently primary, and in those complicating infectious and other diseases, the prognosis is good. The danger is chiefly from the nephritis which follows or complicates the process. In cases depending upon local conditions, the prognosis will depend upon the nature of the exciting cause. Here, also, the principal danger is from nephritis. If calculi are present and if pyonephrosis occurs, the patient may die from exhaustion before a serious degree of nephritis has developed.

**Treatment.**—Water should be given freely, and alkalies up to the point of neutralising the excessive acidity of the urine. In infants, from fifteen to twenty-five grains of the citrate of potash are required daily for this purpose. If the urine is alkaline, benzoic acid may be used in the same doses. The most widely used remedy is hexamethylenamine (urotropin), which may be given in doses of one or two grains every three hours to an infant of a year, and proportionate doses to older children. I have seen it used in large and small doses in cases of acute pyelitis, but have not been convinced of its value, most cases promptly recovering without it. Occasionally pyelitis is very resistant to any form of treatment, the exacerbations and remissions continuing for many weeks. For such obstinate cases vaccines, preferably the autogenous variety, should be tried. Striking benefit has sometimes followed their use. If calculi are present the same treatment is indicated. Surgical interference is called for if pyonephrosis develops, or if the disease is evidently unilateral and the kidney is seriously involved. The advisability of surgical interference will depend upon the clearness of diagnosis and the severity of the symptoms.

#### RENAL CALCULI.

Small renal calculi are very common in infancy. In the autopsy room we frequently see, on opening the kidneys of young infants, fine brown granules in the pelvis and calices, and occasionally a calculus as large as a small pea is found. They are usually composed of uric acid. Only once in over one thousand autopsies of which I have records, was a stone of any considerable size seen in an infant. In this case it was an inch in length and half an inch wide. It is surprising that these are so rare, when we consider how very frequently the minute calculi are met with. The probable explanation is, that the majority of them are dissolved or washed down into the bladder and passed *per urethram* because of the fluid diet of the first two years. The granular deposits are usually lodged in the pelvis of the kidney, and are generally seen upon both sides. With the larger collections there is often a slight catarrhal pyelitis.

**Symptoms.**—The small deposits give no symptoms, and even quite large calculi may be found at autopsy where no indication of their presence had existed during life, as in the case above mentioned. In some cases symptoms are produced which resemble those of renal calculi in the adult. In infants less definite symptoms are often passed over as merely intestinal colic.

In well-marked cases in older children there is tenderness, pain localised over the affected kidney, or radiating to the bladder, the perinaeum, and even the opposite kidney, and there may be irritation and retraction

of the testicle. The urine may show, especially after exercise, a trace of blood; there may be the added symptoms of pyelitis, with some fever, localised tenderness, and the appearance in the urine of pus and epithelial cells from the pelvis of the kidney.

Renal colic is produced when a stone of any considerable size passes from the kidney to the bladder. It is characterised by symptoms similar to those seen in the adult. There are sudden attacks of severe sickening pain in the loins, shooting down the thigh or to the testicle. There may be vomiting and even collapse. The urine is passed frequently, in small quantities, and contains blood. The symptoms quickly subside when the stone reaches the bladder. The calculus may sometimes become impacted in the ureter and give rise to hydronephrosis or pyonephrosis, which soon becomes pyelo-nephritis.

The existence of small calculi may be suspected from the symptoms above mentioned; the diagnosis is made positive by the appearance of gravel in the urine. The use of the Röntgen rays is of service in recognising even small calculi.

**Treatment.**—The only medical treatment consists in a fluid diet, the free use of alkaline mineral waters, and a sufficient quantity of some drug to render the urine alkaline. Such measures will relieve only the milder conditions. With larger calculi and more marked symptoms, a surgical operation should be considered and should be urged in proportion to the severity of the symptoms and the clearness of the diagnosis. If calculous pyelitis exists, it is certain sooner or later to lead to serious nephritis, and it is only a question of time when the kidney will be disabled. The same is true of hydronephrosis from the impaction of a calculus in the ureter. Aldibert has collected four cases of nephrectomy in children for renal calculi in which the kidney was healthy, with three recoveries and one death from shock. In nine cases of operation for calculous pyonephrosis, there were six recoveries and three deaths. The earlier the operation the greater the chances of success, because of the better condition of the other kidney. Although the continued use of water and the so-called solvents may relieve some of the symptoms, it is very questionable whether they do more.

#### TRAUMATIC HYDRONEPHROSIS.

In addition to the hydronephrosis which results from congenital malformations and from the impaction of calculi, a form is occasionally seen following severe injury to the kidney. The pathology of hydronephrosis in these cases is not well understood. After the early symptoms of traumatism have subsided, there develops in from two weeks to two months a tumour in the region of the kidney, which may reach a considerable size and present all the ordinary characteristics of hydro-

nephrosis arising from other causes. This tumour may disappear spontaneously, or it may increase in size and demand surgical intervention for its cure. In seventeen cases which Aldibert has collected there was only one of spontaneous recovery; aspiration was done in seven cases, with six cures and one death; incision with or without nephrectomy was practised in nine cases, with seven recoveries and two deaths.

#### PERINEPHRITIS.

This consists in an inflammation in the cellular tissue surrounding the kidney, which may terminate in resolution or in suppuration. It is not of very uncommon occurrence, and is of importance chiefly from the frequency with which it is confounded with disease of the hip or spine. Perinephritis may be secondary to suppurative processes in the kidney itself, whether from calculi or tuberculous deposits, or it may be primary. In children the latter is the common form. Primary perinephritis is attributed to traumatism, cold, or exposure, or it may develop without assignable cause. It usually runs an acute or subacute course; very rarely it may be chronic.

For the clinical picture of this disease I am chiefly indebted to a paper by Gibney, who has published a report of twenty-eight cases of primary perinephritis in children. The ages of these patients were between one and a half and fifteen years, the majority being between three and six years. The two sides and the two sexes were about equally affected. About one-third of the cases were clearly traceable to traumatism; in the others no adequate exciting cause could be discovered. The majority of the cases were referred to the hospital with the diagnosis of hip-joint disease or caries of the spine. Resolution followed in twelve of these cases, and sixteen terminated in suppuration.

When abscess forms, it usually burrows between the lumbar muscles and comes to the surface posteriorly near the middle of the ilio-costal space; it may burrow forward between the abdominal muscles and point just above Poupart's ligament; very rarely it may follow the psoas muscle and appear at the upper and inner aspect of the thigh, like an ordinary psoas abscess; or it may open into the peritoneal cavity.

**Symptoms.**—The onset of acute perinephritis may be quite abrupt, with chill, fever, and localised pain; or it may be gradual, with stiffness of the spine, lameness referred to the hip, and deformity due to contraction of the flexors of the thigh. The pain is usually felt in the loin, but may be referred to the groin, to the inner side of the thigh, or to the knee. It is often severe, and increased by using the limb. It is in most cases accompanied by localised tenderness in the neighbourhood of the kidney. There is lameness upon the affected side, which may come on gradually, being sometimes referred to the hip and sometimes to the

spine. These symptoms often develop slowly in the course of two or three weeks. They are usually accompanied by a slight elevation of temperature. In the most acute cases the temperature is high ( $102^{\circ}$  to  $104^{\circ}$  F.), and prostration severe.

As the disease progresses, fever is a constant symptom, the temperature usually varying between  $101^{\circ}$  and  $103^{\circ}$  F. There is in most cases increasing deformity, and finally the patient may be unable to walk at all. On examination at the height of the disease, there is found in a typical case a deviation of the spine with the concavity toward the affected side; the thigh may be held flexed to a right angle; passive extension is resisted and causes pain, although all the other movements at the hip joint are normal. In the lumbar region there is tenderness, and there may be an area of infiltration filling the ilio-costal space. At first this is only appreciable by percussion, but later a distinct tumour is present. In addition to the tumour in the usual region, there is sometimes one at the upper and inner aspect of the thigh, owing to a burrowing of pus, and the sacs may communicate.

Lameness, pain, deformity, and fever sometimes exist for two or three weeks before any tumour can be made out. The constitutional symptoms are often severe. The size of the abscess is sometimes very great. In one case I saw it extend from the spine to the median line in front, and from the crest of the ilium nearly to the free border of the ribs. The amount of pus varies from a few ounces to two or three pints. Urinary symptoms are sometimes wanting; at other times there is increased frequency of micturition, accompanied by pain from an irritation referred to the bladder. The urine may contain pus from a complicating pyelitis. In only one of Gibney's cases was this present. It developed in the fourth week, and the case recovered.

The duration of the disease in the acute cases varies from three to eight weeks; in the subacute it may be five or six months. When suppuration occurs the symptoms subside quite rapidly after the pus has been evacuated, and recovery is complete. When resolution takes place, there is a gradual subsidence of the symptoms, and often some stiffness of the thigh, with slight lameness for several months. In the series of cases above referred to, sixty-five per cent recovered completely in three months.

**Diagnosis.**—In many cases a diagnosis of hip-joint disease is made, but that disease develops more insidiously, is very much more chronic, and rarely produces so great deformity in a year as is often seen in perinephritis in two or three weeks; abscess is infrequent during the first year of the disease. In perinephritis, on the other hand, we have a tolerably acute onset, sometimes with chill, fever, marked lameness, and deformity, developing in two or three weeks; abscess often forms in a month, and complete and permanent recovery usually follows after a

few months at most; the deformity is due solely to flexion of the thigh; all other movements at the hip may be free, and joint tenderness is absent. Psoas abscess from Pott's disease may cause deformity, tumour, and lameness similar to that seen in perinephritis, but on examination there is found the angular prominence and other signs of disease of the lumbar vertebræ. In cases of doubt the tuberculin test may give important aid in diagnosis.

**Prognosis.**—Primary perinephritis in children almost invariably terminates in complete recovery. Of the twenty-eight cases referred to, and eight subsequently observed by Gibney, all recovered perfectly. The only condition likely to prove fatal is rupture of the abscess into the peritoneal cavity.

**Treatment.**—The patient should be put to bed and kept as quiet as possible throughout the attack. In the early stage, hot fomentations or an ice-bag should be applied over the affected side; heat is generally to be preferred. Abscesses should be opened early, to prevent burrowing and the danger of a possible rupture into the peritoneal cavity.

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### CHAPTER III.

#### *DISEASES OF THE GENITAL ORGANS.*

##### MALFORMATIONS.

**Adherent Prepuce.**—This condition is sometimes called false phimosis. It is so constantly present that it can hardly be regarded as a malformation. It is, however, a condition needing attention in every male infant. The prepuce should be forcibly retracted so as to expose the glans completely. The smegma should then be washed away, the glans covered with a drop of oil, and the skin drawn forward. This should be repeated daily until there is no disposition to a recurrence of the adhesions.

**Phimosis.**—This is such a narrowing of the prepuce that it can not be retracted over the glans. The degree of phimosis varies greatly. In very rare cases there is no preputial opening. In other cases the orifice is so small that no part of the glans can be exposed, and there is obstruction to the outflow of urine; but usually a small part of the glans can be seen. Phimosis may be complicated by an elongated prepuce (hypertrophic phimosis), and the elongation may exist without any narrowing of the orifice, although this is usually present to some degree.

The presence of phimosis makes cleanliness impossible in many cases, and want of cleanliness leads to infection and to balanitis. This is quite frequent, even in infants. It may be complicated by urethritis, and even



by cystitis. Another consequence of the straining induced by phimosi is hernia, which may be either inguinal or umbilical. To cure the hernia is often impossible, unless the phimosi is relieved. Reflex symptoms may come from preputial adhesions as well as from phimosi. The hyperæsthetic condition and the resulting pruritus cause frequent priapism, and are among the common causes of masturbation. Phimosi may produce other nervous symptoms, such as insomnia, night terrors, etc. It often causes frequent micturition, dysuria, and, in fact, most of the symptoms of stone in the bladder. It sometimes leads to vesical spasm and retention of urine, but more frequently to nocturnal incontinence.

The list of reflex phenomena which have been ascribed to phimosi is a long one, and includes most of the functional nervous diseases of childhood. There has been in the past a disposition on the part of some to attribute nearly all the nervous disturbances of boyhood to phimosi, and an exaggerated importance has certainly been attached to this condition. Still, in a delicate, anæmic child with unstable nervous centres, phimosi is capable of giving rise to nervous symptoms of a serious character. It is an important etiological factor in many neuroses, and one which should not be overlooked. On the other hand, a very marked degree of phimosi often exists in healthy children without producing any symptoms whatever.

*Treatment.*—Every case of phimosi should receive attention in infancy. Often very little treatment is needed; but trouble is likely to come sooner or later if it is neglected. When there is a very long prepuce with phimosi, the operation of circumcision should in my opinion be done, even when the degree of phimosi is slight. Many cases of phimosi in which the prepuce is not long can be relieved by stretching. If no part of the glans can be exposed, the simplest plan is to slit up the dorsum of the prepuce with a pair of scissors and forcibly break up the adhesions. The corners of the flaps thus made can then be snipped off and one stitch inserted on either side. In the case of obscure nervous symptoms in older boys, the condition of the prepuce should be examined and the same rules of treatment applied. In cases of hernia, or prolapsus ani, when phimosi is present it should be relieved.

**Hypospadias.**—In this condition the urethra is not continued to the tip of the penis, but opens on the inferior surface some distance back, being represented in front of this only by a shallow furrow. In more severe cases there is a deep fissure which divides the scrotum, and sometimes even the perinæum. Into this fissure the urethra opens. This is a condition likely to be mistaken for that of hermaphroditism, especially as the testicles are frequently in the abdominal cavity.

**Epispadias.**—This is a condition in which the urethra opens on the dorsal surface of the penis. It is much less frequent than hypospadias. There may be simply a division of the glans, or the fissure may extend

the whole length of the organ and be complicated by exstrophy of the bladder.

**Exstrophy of the Bladder.**—In the complete form there is a median fissure from the umbilicus to the tip of the penis. It includes the anterior abdominal wall, the pelvic bones, and the urethra. The bones are entirely separated at the symphysis, or connected behind the bladder by a fibrous band. The hypogastric region is occupied by a red, mucous surface, slightly corrugated, which is all there is of the bladder. In the lower lateral portions of the red mucous membrane two slightly rounded elevations are seen, from which urine oozes. These are the openings of the ureters. The penis is short, and presents a shallow furrow on its dorsal surface. The testes are often in the abdominal cavity.

An analogous deformity is sometimes seen in girls. There is a division of the clitoris and the labia minora and majora. The fissure may be so deep as to reach nearly to the anus. The vagina is usually absent. The rectum may open into the prolapsed bladder.

All these deformities are compatible with long life. In most of them the individual is incapable of procreation. In exstrophy of the bladder, whether complete or partial, patients are a nuisance to themselves and to all about them. It is almost impossible to prevent the clothing from being soaked with urine, which gives everything connected with the patient a strong ammoniacal odour. The skin is often excoriated. Operation for the relief of these cases should, I think, always be undertaken. The operation to be recommended is the transplantation of the ureters into some part of the large intestine, usually the rectum. The results are often most surprising. The rectum soon becomes tolerant of the urine, holds it for hours without difficulty and evacuates it without discomfort. Ascending infection of the kidney seldom occurs.

**Undescended Testicle—Cryptorchidism.**—In foetal life the testes are situated in the abdominal cavity below the kidneys. They usually descend into the scrotum during the ninth month, but in children born at term the testicles may be in the inguinal canal, or even in the abdomen. The former condition is quite frequent, being present in fully ten per cent of all male children. In most of these the descent takes place without difficulty during the first weeks of life, and causes no symptoms. In others the condition may persist. Spontaneous descent may take place at any time before puberty, the chances, however, steadily lessening as age advances. When in the inguinal canal, on account of its exposed situation, the testicle may be injured, or become painful and tender as puberty approaches. In any abnormal position it probably will not develop properly, and may remain without function, but interference with the development of the body is rare. Hernia is a frequent complication.

When in the inguinal canal, descent of the testicle may sometimes be facilitated by manipulation. If the condition is unilateral, operation is

unnecessary except for relief of pain. If it is double, operation should be performed before puberty, preferably in the eleventh or twelfth year. Transplantation into the scrotum is at this time simple, and usually successful. Should pain be persistent, and transplantation impossible, the testicle may be replaced in the abdominal cavity. Removal is indicated only when degeneration has taken place.

With the exceptions already mentioned, deformities of the female genitals belong rather to gynaecology than to pædiatrics, since they are chiefly of the internal organs, and do not usually give symptoms before puberty.

#### DISEASES OF THE MALE GENITALS.

**Balanitis.**—Balanitis, or inflammation of the prepuce, is one of the results of phimosis. It may follow decomposition of the smegma, infection of the mucous membrane, injury, or masturbation. The parts are swollen, œdematous, red, painful, and sometimes bathed in pus. Retraction of the prepuce is impossible. Under proper treatment the inflammation usually subsides in two or three days, but there may be some discharge for a considerable time. Abscess may follow, and even gangrene of the prepuce. The most severe cases are likely to be complicated by anterior urethritis. I have frequently seen erysipelas start from balanitis, and occasionally diphtheria occurs here.

The object of treatment is to remove the irritating and infectious material lodged beneath the foreskin. This may be quite difficult. It is best accomplished by syringing with a 1-5,000 bichloride solution, and the constant application of a wet antiseptic dressing. Ice is often useful when the œdema is great. It is sometimes necessary to slit up the prepuce before the parts can be thoroughly cleansed, and in severe cases this is often the quickest method of cure. Circumcision should not be done during an attack.

**Urethritis.**—This, like the same disease in females, may be simple or specific. Both forms are less frequent in little boys than in the other sex. In simple urethritis the inflammation usually affects only the anterior part of the canal, the fossa navicularis. There is a slight discharge of pus, and sometimes pain on micturition. The most frequent cause is want of cleanliness.

Gonorrhœal inflammation is more common. This occurs even in infants, but most of the cases are in those over seven years old. The usual cause is direct contagion. The symptoms are more severe than in the simple form, and resemble the same disease in the adult, with the exception that constitutional symptoms are usually absent. A microscopical examination of the discharge is the only positive means of diagnosis between the two varieties. In these cases it reveals the gonococcus in great numbers. Conjunctivitis and arthritis are seen as complications,

just as in the female. Orchitis is very rare, but balanitis and bubo are not infrequent. Poynter has reported a case in a boy of three years, who, when five years old, required treatment for a urethral stricture. He was infected by a nurse.

The first thing in the treatment is always to keep the parts covered, otherwise the infection is almost certain to be carried by the hands to other mucous membranes, usually the conjunctiva. In other respects the treatment is the same as in the adult.

**Hydrocele.**—Hydrocele consists in an accumulation of serum in some part of the serous pouch brought down by the testicle in its descent. In infants it is usually due to the imperfect closure of this pouch at some point, where a fluid accumulation occurs. Four varieties of hydrocele are met with in young children:

1. *Congenital Hydrocele.*—In this the condition is a congenital one, although the tumour is not necessarily present at birth. The tunica vaginalis communicates with the general peritoneal cavity. There is present an elongated tumour, extending from the bottom of the scrotum throughout the whole length of the cord. The tumour is reducible, sometimes spontaneously by position, sometimes, when the opening is smaller, only by pressure. It reduces slowly, without gurgling, never going back *en masse* like a hernia. The tumour is translucent, and is flat on percussion. The testicle is above and posterior, and usually indistinctly felt. Congenital hydrocele may be complicated by hernia.

2. *Hydrocele of the Tunica Vaginalis with the Canal Closed.*—In this form the accumulation of fluid is in the scrotum, communication with the peritoneal cavity having been entirely cut off by the complete obliteration of this pouch in the canal in the normal way. This is one of the most frequent forms. It gives rise to an oval or pear-shaped tumour, quite tense and firm, usually about two inches in length. The cord is distinctly felt above it, the testicle is behind and somewhat above it, and not always felt very distinctly. This variety gives translucency and the usual elastic feeling of a hydrocele.

3. *Hydrocele of the Cord.*—This is one of the rare forms. The serous pouch which accompanies the spermatic cord is open above, and communicates with the peritoneal cavity; but below it is closed. The scrotum is normal, and the testicle is in its usual position. The tumour is small, elongated, and reducible, and entirely above the scrotum. Usually it stops at some point in the inguinal canal. This hydrocele also may be complicated by hernia. The diagnostic points are the same as in the form first mentioned.

4. *Encysted Hydrocele of the Cord.*—The peritoneal pouch of the cord in this variety is closed for some distance above, and again below, but somewhere in its course it is open, and here the fluid accumulates in the form of a cyst. When small it resembles an undescended testicle;

but on examination this organ is found below and in its normal position. When in the canal, it is often mistaken for a lymph gland, sometimes for a small hernia. The tumour is usually about the size of an almond. It is elastic and irreducible, and translucent like the other varieties.

*Treatment of Hydrocele.*—In the congenital form the application of a truss will sometimes cause obliteration of the canal, so as to shut off the hydrocele sac from the general peritoneal cavity. It is subsequently managed like an ordinary hydrocele of the tunica vaginalis. In infants and young children it is rare that active operative measures are called for in any variety of hydrocele, as these usually tend to disappear spontaneously in the course of a few months. Iodine may be applied locally over a hydrocele of the cord, but should not be applied to the scrotum. Some cases are cured by a simple puncture with a needle, allowing the fluid to drain off into the cellular tissue of the scrotum from which it is absorbed; others by a single aspiration with a hypodermic syringe. It is seldom necessary to resort to the injection of irritants like iodine or carbolic acid, but they may be used if the fluid returns after repeated aspirations.

#### DISEASES OF THE FEMALE GENITALS.

##### *Vaginitis.*

This is a catarrhal inflammation usually affecting only the vaginal mucous membrane, but may involve the urethra, bladder, and, in older girls, the lining membrane of the uterus, the tubes, and even the peritonæum. It may be either simple or specific (gonorrhœal); the purulent form is almost invariably specific.

*Simple Vaginal Catarrh.*—This may be seen at any age, even in infancy, but is most frequent after the second year. It occurs especially in girls suffering from malnutrition and anæmia, and whose personal cleanliness is neglected. It may follow any of the infectious diseases, particularly measles. It sometimes complicates varicella with a local lesion in the vagina. It may be traumatic, as from attempted rape or the introduction of foreign bodies. Other causes are pinworms and scabies. It is sometimes the cause, sometimes the result of masturbation.

*Symptoms.*—The disease generally begins as a subacute catarrhal inflammation, the discharge being the first, and in mild cases the only symptom. It is of a white or yellowish white colour and not very abundant. If the parts are not kept clean the odour of the discharge is quite foul. In severe cases the discharge is abundant, and may excoriate the skin of the labia and thighs. The mucous membrane is swollen and red, but there is only a moderate secretion. Microscopical examination of the discharge shows bacteria in large numbers and of many varieties, but they are chiefly the ordinary cocci. With proper treatment and in children who are in good general condition, the disease usually lasts from

one to three weeks; or, under unfavourable conditions, there may be a persistent leucorrhœal discharge for a longer time.

**Gonococcus Vaginitis.**—So far from being rare, as was once thought, this disease has been shown by recent observations to be exceedingly common among girls of all ages, even young infants. It is especially in hospitals and other institutions that it is seen, and here it must be considered one of the most frequent and most troublesome of house infections. Routine microscopical examinations which I have had made of the vaginal discharges of children in various institutions usually revealed the existence of gonococcus vaginitis, often in a mild form, in from two to ten per cent of the inmates. Epidemics in institutions are exceedingly common and very difficult to control. Only one who has experienced such epidemics can appreciate what a scourge vaginitis may become. No less than four such epidemics were observed in the Babies' Hospital between the years 1899 and 1904. During this period 273 cases were observed in this institution.<sup>1</sup> Gonococcus vaginitis often exists in day-nurseries or homes for foundlings, as well as in general hospitals and asylums for older children. In out-patient practice, and among the poor in tenements, cases are constantly seen, and even among the well-to-do this disease is by no means rare. From the manner in which it is contracted, it should not, in young children, be considered a venereal disease.

In institutions, gonococcus vaginitis can generally be traced to some child admitted with an acute form of the disease. Before the condition is recognised and the patient quarantined, an entire ward or dormitory may be infected, and a local epidemic may be the result, and once well under way this may last for months.

In infants and young children the disease is seldom acquired by sexual contact, but most frequently through the medium of napkins. Other possible means of infection are towels, sponges, wash-cloths, underclothing, bed-linen, thermometers, syringes, bath-tubs, bath water, or the hands of the nurse. Even when the most careful attention has been given to these matters, I have frequently seen ward epidemics continue unabated. Atmospheric infection seems unlikely. The most probable explanation under these circumstances is that the disease is spread by nurses in washing, feeding, dressing, or bathing children, but especially in the changing of napkins. In many cases it was found impossible to check epidemics until both the patients and their attendants were quarantined.

In girls from six to twelve years old other means of contagion must be considered. This may be by direct contact, manual or sexual, or sleep-

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<sup>1</sup> See author's article on *Gonococcus Infections in Institutions*, *New York Medical Journal*, March, 1905.

ing with parents or others who may have the disease. Pott found in ninety per cent of his cases that the mother had a leucorrhœal discharge. The mode of contagion may be difficult to trace, but this fact should cast no doubt upon the diagnosis.

*Symptoms.*—In infants and young children, in the mild cases, the disease is limited to the mucous membrane of the vagina. There is a moderate yellow discharge which, by microscopical examination, contains pus cells and gonococci. There is little redness and no symptoms of discomfort. In more severe cases the discharge is copious, often thick and of a yellow or yellowish-green colour. It may be tinged with blood from slight erosions. It often causes excoriation of the labia or thighs. In some cases the urethra is involved, causing frequent, painful micturition. The inflammation may extend to the bladder, but seldom or never at this age to the mucous membrane of the uterus. Occasionally the mucous membrane of the rectum is involved. The symptoms are chiefly local, but there may be a slight rise of temperature to 100° or 101° F. during the period of most acute inflammation.

In girls past the age of six or seven years, the symptoms resemble those of the adult: copious secretion, the formation of crusts on the labia, frequent, painful micturition from involvement of the bladder and urethra, and difficulty in locomotion. There may be slight fever and general malaise. The inflammation may extend to the lining membrane of the uterus and, through the Fallopian tubes, to the pelvic peritonæum. Sanger has reported such a case in a child of three years. The endometritis may be demonstrated by the use of a small speculum, by which the discharge may be seen coming from the cervix. Swelling, and very rarely suppuration, of the inguinal glands may take place.

A positive diagnosis between simple and gonococcus vaginitis can be made with certainty only by a microscopical examination of the discharge, though in default of such examination an abundant purulent catarrh may be assumed to be due to the gonococcus. In simple catarrh the discharge is made up of epithelial and pus cells, with quite a wide variety of bacterial forms, chiefly cocci and bacilli, occasionally a few diplococci. In gonococcus vaginitis the gonococci are found in large numbers, and are usually the only bacteria present. To be diagnostic, they should be demonstrated within the pus cells as well as outside them. The gonococcus decolourises when stained by Gram's method, which fact distinguishes it from the other organisms likely to be present in the vagina. The staining is quite as diagnostic as the cultural characteristics of this organism. Cases of vaginitis are to be regarded as suspicious if pus is found and few organisms are detected; in such conditions subsequent examination usually reveals the gonococcus. In my hospital experience the gonococcus cases have outnumbered the simple purulent forms, fully ten to one.

In infants, when the amount of discharge is small and likely to be overlooked, it is an advantage to apply between the labia a fold of gauze upon which the yellow stain of a purulent discharge is readily noticed.

*Gonococcus vaginitis* may be complicated by conjunctivitis, arthritis, endo- or pericarditis, peritonitis, and proctitis. Conjunctivitis is the most frequent, the infection usually being carried by the hands. *Gonococcus arthritis* is not uncommon even in young infants. It is usually a multiple arthritis, with the constitutional symptoms of pyæmia. The wrist, ankle, knee and elbow, and small joints of the fingers and toes are most frequently involved. These cases are considered more fully in the chapter on Acute Arthritis in Infants.

The diagnosis in all the complicating conditions is based upon the presence of the gonococcus.

*Prophylaxis*.—The highly contagious character of *gonococcus vaginitis* makes it imperative that such cases should not be received into the same ward or dormitory with other children. Only in this way can house epidemics be prevented. Cases which are mild should be excluded, as well as those which are severe. The only effective measure is to make the microscopical examination of vaginal discharges of children admitted to an institution as much a matter of routine as the taking of throat cultures if there is a tonsillar exudate. Cases showing the gonococcus should be quarantined or excluded. When there are a great many admissions every month, a case occasionally escapes detection. The rule which we have followed in the Babies' Hospital has been to make not only an examination on admission, but routine examinations of all patients at stated intervals, always once and sometimes twice a week. Only by this means has it at times been possible to eradicate the disease.

The attendants, both day and night nurses, as well as the children, should be quarantined. Napkins, underclothing, and sheets from the beds of infected children, also towels and wash-cloths, should not go into the common laundry, but should be first soaked in a strong solution of carbolic acid, and afterward boiled. In wards or institutions where cases have occurred washable napkins should be discontinued and old muslin and absorbent cotton used in their place. These are to be destroyed after using. All articles connected with the children's toilet, also syringes, thermometers, etc., should be carefully disinfected. But often this is not enough. Separate articles should be furnished for each child. The organism is one that is fairly easy to kill, and if proper precautions are taken epidemics may be prevented. The essential measure is a prompt recognition and isolation of the first case in the hospital. Quarantine should continue not only until the catarrhal inflammation has subsided and the organism has disappeared, as shown by a single negative microscopical examination, but for a considerable time longer, since a slight



discharge containing a few organisms may remain for weeks after the case is considered cured. Relapses are very frequent.

*Treatment.*—Cases of simple vaginal catarrh should be irrigated twice daily with a warm saturated solution of boric acid or 1 to 5,000 bichloride. Cleanliness should be secured by frequent bathing and the skin protected by ointments. In more severe cases, astringent injections, such as sulphate of zinc and tannic acid (of each one drachm to a pint of water) should be used, or protargol applied in solutions of from one to five per cent strength. The general health should be built up by iron, cod-liver oil, and other tonics.

In gonococcus vaginitis more energetic treatment is necessary. Every child should wear a napkin, to prevent carrying the infection to the eyes by means of the hands. Irrigations should be used at least twice a day, and stronger antiseptics employed than in the simple cases. The best are protargol, in solutions from one to ten per cent strength, and argyrol, in solutions from five to twenty-five per cent strength. Applications should be made with a cotton swab; the same substances may be used in the form of suppositories, or the vagina may be packed with gauze wet in these solutions. The closest attention to cleanliness is required in all cases. The course of the disease is very tedious; many weeks, and often months, may be required for a cure. On the whole, treatment is very unsatisfactory on account of the difficulties in the way of making thorough local applications. When the disease involves the bladder and urethra, the same general measures as in adults are indicated.

The precise place and value of vaccines in the treatment of gonococcus vaginitis is undetermined, reported results with this method being far from uniform. When practicable I believe that they should be given a trial in all chronic or specially resistant cases. I have personally seen a few brilliant results from their use. I have generally employed stock vaccines made from many strains of the gonococcus. Dosage is still a matter of much uncertainty. Fifty to seventy-five millions may be used every four or five days until five or six doses have been given. I have seen no unfavourable symptoms in any case.

#### *Gangrenous Vulvitis (Noma).*

This is the same process as that seen in the mouth and known as cancrum oris. It usually follows one of the infectious diseases, most frequently measles, occurring in patients whose general vitality has been greatly reduced. There is first noticed a tense, brawny induration, the skin being shiny and swollen over a circumscribed area. In the centre of this there soon appears, usually upon one of the labia majora, a dark, circumscribed spot. Day by day the gangrenous area advances, preceded by the induration. It may involve the whole labium, extending even to the mons veneris and the perinæum. These cases are generally fatal.

If recovery takes place, it is with considerable deformity of the parts in consequence of the extensive sloughing and cicatrisation. As sequelæ, there may be fistulæ, stenosis, or atresia of the vagina. The only radical treatment is early excision, and the application of the actual cautery, carbolic or nitric acid.

## CHAPTER IV.

### DISEASES OF THE BLADDER.

#### ENURESIS.

*(Incontinence of Urine; Bed-wetting.)*

ENURESIS may be due to some malformation of the genital tract, such as an abnormal opening of the bladder into the vagina, to extroversion of the bladder, or to the persistence of the urachus; in the latter case the urine is discharged from the umbilicus. It also occurs in organic diseases of the central nervous system, such as idiocy, cerebral palsy, acute meningitis, tumours of the brain, certain forms of myelitis, and in injuries of the cord. In many of these conditions there is associated incontinence of fæces. Both of the groups of cases mentioned are quite distinct from the ordinary form of incontinence of urine which is seen in childhood. The latter is to be regarded as a neurosis, and is the only variety which will be considered here.

It is in many cases possible to teach infants to control the evacuation of the bladder before the end of the first year; usually, however, control is not acquired even during waking hours until some time during the second year, and in some healthy infants not before the end of the second year. The time depends very much upon the training. If a child during its third year can not control the evacuation of the bladder during its waking hours, incontinence may be said to exist.

**Etiology.**—Incontinence of urine may be due to a continuance of the infantile condition, to anything which increases the irritability of the spinal centre, or which interferes with the cerebral control over this centre, or to anything which increases the irritability of the terminal filaments of the vesical nerves or of those in the neighbourhood. The causes of incontinence thus may be in the central nervous system, in the urine, in the bladder, or in any of the adjacent organs.

The causes relating to the central nervous system are in the main those of the other neuroses of childhood; these are anæmia, malnutrition, an inherited nervous constitution, or a condition of extreme nervousness or neurasthenia, the result of the child's surroundings. In such cases incontinence is often associated with chorea, epilepsy, hysteria, headaches, neuralgia, and other nervous symptoms. In these conditions there may be not only an increased irritability of the nerve centres, but also of the

peripheral nerves, accompanied by loss of tone of the vesical sphincter. A similar condition may exist with almost any form of acute illness, usually, however, being only temporary.

Incontinence may be caused either by a highly acid, concentrated urine when an insufficient amount of fluid is taken, or by the opposite condition, where, owing to the drinking of a large quantity of water, often only a matter of habit, the amount of urine is very greatly increased and passed at frequent intervals.

In the bladder itself, cystitis and vesical calculus, although infrequent, should not be overlooked as possible causes. In a few cases, where incontinence has existed a long time, the bladder becomes so contracted that it will hold only an ounce or two of urine. This condition, although not the primary cause of enuresis, may be enough to continue it.

Local irritation in the neighbouring organs may be due to adherent prepuce, balanitis, phimosis, or to a narrow meatus. All of these conditions are frequently associated with incontinence. Rectal irritation may be due to pinworms, anal fissure, or rectal polypus; and vaginal irritation to vulvo-vaginitis or adherent clitoris; but these are rarely the only cause. Often we have incontinence as the result of a combination of several causes, no one of which alone would have been sufficient to produce it. Thus, in a healthy child phimosis may give rise to no symptoms, while in one who is anæmic or neurasthenic it may produce enough local irritation to cause incontinence. In many cases heredity seems to be a factor of some importance, parents often having suffered in their childhood from the same condition; quite frequently two and sometimes even three children in the same family are affected. In many cases the condition seems to be mainly the result of habit, and in all cases habit is a potent factor in continuing the incontinence, sometimes after the original exciting cause has been removed. Frequently no adequate cause can be found. Both sexes are about equally liable to enuresis, and it may be seen in all ages up to puberty.

**Symptoms.**—Enuresis may be nocturnal or diurnal, or both. Of 184 cases, 73 were nocturnal, 9 diurnal, and 102 were both nocturnal and diurnal. Cases differ greatly in severity. Incontinence may be habitual, occurring every night, often several times during the night, and frequently during the day; or it may be only occasional under the influence of some special exciting cause, when it continues a few days or weeks until the cause is removed. In a considerable number of cases, the condition lasts from infancy until the sixth or seventh year. It may even continue until puberty; but it generally ceases at that period, unless its cause is mechanical or depends upon some organic disease of the brain or cord. In ordinary enuresis there is never dribbling of the urine, but usually a contraction of the walls of the bladder follows almost immediately upon the desire before the patient can make his wants known or reach a con-

venient place for micturition. At night the same thing may occur without wakening the child, the contraction being of purely reflex origin.

**Prognosis.**—The condition is usually hopeless when it depends upon organic disease of the brain and cord; also in cases due to malformation, unless these are amenable to surgical treatment. In the ordinary cases seen, the prognosis depends upon the age of the child, the duration of the symptom, and the nature of the exciting cause. In children of from three to five years a cure can in many cases be accomplished with proper management. Those who are older are much less amenable to treatment, especially if the condition has persisted since infancy; but if the incontinence has begun after seven or eight years of age and lasted a few weeks or months, the outlook is much more encouraging. When some cause can be discovered which can be removed, the prognosis is better than if none can be found. There are, however, some cases in which no other cause than habit can be discovered which resist all treatment, the condition finally ceasing spontaneously at or a little before puberty; in very few does it continue beyond this period.

**Treatment.**—The first indication is to remove the cause, when one can be found. If there are preputial adhesions, they should be broken up and irritating smegma removed. If phimosis is present, it should be relieved by stretching or circumcision. If stone in the bladder is suspected, as it should be when the incontinence is worse by day and accompanied by straining and painful spasm of the bladder, the patient should be sounded for stone. Pinworms in the rectum should receive the appropriate treatment by injections. While the local conditions mentioned should always be attended to, the fact remains that few cases are cured simply by relieving them, except those due to vesical calculi. The explanation of this is that habit is a very important factor in keeping up incontinence where it has existed a long time.

A concentrated urine of high acidity with deposits of uric acid is an indication for alkalies and the free use of all fluids, especially water. On the other hand, when there is passed a large quantity of urine with low specific gravity, the amount of water and other fluids should be restricted. During the night water should be forbidden, and the amount given in the latter part of the day greatly reduced. In these cases the incontinence is often simply the result of the polyuria, which in turn depends upon polydipsia.

In most cases the condition is a nervous habit, and usually associated with other habits which indicate an unstable or highly susceptible nervous system. It is therefore of the greatest importance that a proper general régime should be instituted and enforced. Care should be taken to secure for the child a simple, natural life, preferably in the country. There should be no overtaking of the nervous system at home or in school. Every cause of unnatural excitement should be avoided. Early

hours and plenty of sleep must be insisted upon. Certain articles of diet are to be avoided, and coffee, tea, and beer should be absolutely prohibited. Sweets and all highly seasoned food should be very sparingly allowed, or not at all. Although it is believed by many that a diet into which meat enters largely is injurious, from personal experience I have not found the exclusion of meat to be of any advantage. The diet which succeeds best is a simple one composed of milk, vegetables, fruits, meats, and cereals. With most patients who have nocturnal incontinence, it is well to allow fluids freely during the early part of the day, but little or none after 3 or 4 P.M., a dry supper being given just before retiring. The child should be taught to hold his water as long as possible during the day, to accustom the bladder to full distention.

Measures directed toward improving the general muscular and nervous tone are of the greatest importance. It should be remembered that incontinence of urine is a neurosis, depending, like most neuroses of childhood, upon disturbed nutrition. Anæmia, chlorosis, malnutrition, indigestion, and constipation should each receive careful attention. Any local condition, such as adenoid growths of the pharynx, which might serve to increase the general nervous irritability, should be removed. Yet, very few cases are cured by such an operation.

Moral treatment is also important. One should work upon the child's pride and use every possible means to strengthen his will. Punishments, whether corporal or otherwise, do little good, and with most children they are absolutely harmful. With children in whom incontinence is chiefly a matter of habit, I have often found rewards more efficacious than any other means of treatment. One should first find out what it is that the child desires most—a new doll, a bicycle—and allow him to have it if his bed is dry, taking it away if it is wet. A reward of five cents for every dry night sometimes works marvels.

The measures described—removal of local causes, building up of the general health, institution of a proper régime, and mental and moral means—in a very considerable number of cases suffice for a cure. They generally constitute the most important part of the treatment. Drugs are useful as accessories, but alone seldom accomplish a cure, and, on the whole, are disappointing. Of those employed, belladonna is certainly the most effective, but its administration should be continued for a long time. Atropine, either in solution or in tablet form, is the most convenient method of administration. For nocturnal incontinence,  $\frac{1}{100}$  of a grain for each year of the child's age up to seven years is a suitable initial dose. A child of five would thus be taking  $\frac{5}{100}$  of a grain. At first, a single dose should be given at bedtime; after a few days a second dose may be given three or four hours earlier. To push the drug much further than this causes much discomfort and is of doubtful advantage. After the condition is under control, the same dose should

be continued for some time and then reduced, the atropine being given for at least two months in gradually diminishing doses after the incontinence has ceased. This is very important if the cure is to be permanent, as there is a strong tendency to relapse.

Strychnine may be added in cases not yielding to the atropine alone. It is particularly advantageous when there is diurnal as well as nocturnal incontinence, for under these conditions there is usually a lack of tone in the sphincter, as well as increased irritability in the mucous membrane of the bladder. The initial dose for a child of five years should be  $\frac{1}{160}$  of a grain twice daily; this may be gradually increased to  $\frac{1}{80}$  of a grain three times a day; but there is rarely any advantage in pushing it further. Ergot is sometimes useful in conjunction with other drugs, but rarely gives relief when both strychnine and atropine have failed. Some obstinate cases are reported to have been relieved by faradism; the positive pole is attached to a small electrode passed into the rectum and the negative pole applied over the bladder. The sitting should last for ten minutes, and be repeated three times a week. My own experience with this method of treatment has been disappointing. If there is reason to suspect a contracted bladder, as when the incontinence has lasted for years and the bladder will never hold more than an ounce or two of urine, cure is sometimes accomplished by daily distending the organ up to its normal capacity with warm water. A few obstinate cases in older boys which had resisted all other methods of treatment were cured in my clinic by the passage of sounds.

Careful, intelligent, systematic training is a most valuable adjunct to all measures employed for the relief of this very annoying condition.

#### VESICAL SPASM.

This is quite a common condition, and often passes under the name of *genital irritation*. It is characterised by frequent, sometimes by difficult and painful, micturition. It occurs in children of all ages, even in infants, but is especially frequent between the ages of two and five years. This symptom has already been referred to in connection with uric-acid infarctions in very young infants.

The usual cause is the irritation of the bladder by a concentrated, highly acid urine. It often results from cold; it may accompany acute febrile processes, and is sometimes merely a symptom of nervous irritability. The cause may thus be in the bladder or in the urine. It may be accompanied by enuresis, but usually occurs without it. It is sometimes symptomatic of disease in adjacent parts, as in the rectum or the pelvic peritonæum, or it may be associated with inflammation of the vulva or urethra. It is also one of the symptoms of vesical calculus.

The symptoms of vesical spasm are local only. The child passes

water very frequently, often several times an hour. The accompanying pain may be intense, not infrequently sufficient to cause the child to cry out. Often there is pain and severe vesical tenesmus with the passage of only a few drops of urine at a time, but blood is not present. If the condition depends upon the character of the urine, or is only an expression of an extreme vesical irritability, the symptoms are generally of short duration, possibly a day or two. If it depends upon vesical calculus, it may be intermittent. If it is associated with disease of the adjacent pelvic viscera, it is inconstant, and may continue for a considerable period, depending upon the nature of the cause.

The treatment, in the ordinary cases, consists in the administration of an abundance of water, with alkaline diuretics, and either belladonna or hyoscyamus. The tinctures of these may be given in minim doses every two hours to a child of two years.

If the cause is outside the bladder, it should receive appropriate treatment.

#### VESICAL CALCULI.

Vesical calculus is a very rare condition in children in New York. The nucleus of a calculus is usually a renal calculus which has passed the ureter, but has been prevented by its size from going farther. Stone in the bladder is extremely rare in infancy, probably owing to the fluid diet, but it is not infrequent in children from two to ten years of age. The most common variety of calculus at this time is the uric acid.

The symptoms in children are somewhat different from those in adults, and the condition is often overlooked. There is frequently pain upon micturition, especially at the close of the act, which may be felt at the end of the penis or in the perinæum. There may be a sudden stoppage in the flow of urine. The straining often leads to rectal tenesmus and even to prolapse. This complication is so frequent that, in a case of persistent prolapse, stone should always be suspected. Incontinence of urine is a prominent, and often the principal symptom; in many cases it is noticed only during the day. The urinary changes are not generally marked; hæmaturia is rare, and mucus and pus are infrequent and in small quantity. The genital irritation may lead to the habit of masturbation. A stone of any considerable size may often be felt by a bimanual examination, one finger being placed in the rectum and the other hand above the pubes. This is easier in males than in females, but it is not very trustworthy, and not conclusive when it gives a negative result. A positive diagnosis is made only by exploring the bladder with a sound or by the Röntgen ray.

The treatment of calculus is purely surgical.

# SECTION VII.

## DISEASES OF THE NERVOUS SYSTEM.

### CHAPTER I.

#### INTRODUCTORY.

**The Weight of the Brain.**—From ninety-eight observations made in the post-mortem room of the New York Infant Asylum, the following were the average weights noted:

|                        |     |                    |
|------------------------|-----|--------------------|
| At three months .....  | 21  | oz. (602 grammes). |
| At six months .....    | 25½ | " (712 " ).        |
| At twelve months ..... | 32½ | " (916 " ).        |
| At two years .....     | 35  | " (990 " ).        |

The following are the figures given by Boyd and Schäfer:

| Age.                                | Males.  |          | Females. |          |
|-------------------------------------|---------|----------|----------|----------|
|                                     | Ounces. | Grammes. | Ounces.  | Grammes. |
| At birth (full term) .....          | 11½     | 330      | 10       | 283      |
| Under three months .....            | 17½     | 500      | 16       | 450      |
| From three to six months .....      | 21      | 602      | 20       | 560      |
| From six to twelve months .....     | 27      | 776      | 26       | 727      |
| From one to two years .....         | 33      | 941      | 30       | 843      |
| From two to four years .....        | 39      | 1,110    | 35       | 990      |
| From four to seven years .....      | 40      | 1,138    | 40       | 1,135    |
| From seven to fourteen years .....  | 46      | 1,301    | 40½      | 1,154    |
| From fourteen to twenty years ..... | 48½     | 1,374    | 44       | 1,244    |

At birth the weight of the brain to that of the body is nearly 1:8. During infancy and childhood the following is the ratio, according to Bischoff: during the first year, 1:6; the second year, 1:14; the third year, 1:18; at the fourteenth year, 1:15 to 1:25; in adults, 1:43.

**The Spinal Cord.**—The weight of the cord to the weight of the body at birth is 1:500; in adult life it is 1:1500. According to Kölliker, the spinal cord and the vertebral column are the same length until the end of the third month of fœtal life, there being at this time no cauda equina. At the ninth month the lower end of the cord is opposite the third lumbar vertebra; in the adult it is opposite the first.

**Some Peculiarities in the Diseases of the Nervous System in Infancy and Childhood.**—The relatively large size, the rapid growth, and the im-



maturity of the brain and cord during early life, explain much that is peculiar to the nervous diseases of this period.

At this time, apparently trivial causes are enough to produce quite profound nervous impressions, because of the instability of the nervous centres and the greater irritability of the motor, sensory, and vaso-motor nerves. These are conditions which are very much increased by all disturbances of nutrition. These disturbances may be manifold in character, but they lie at the root of very many of the neuroses of early life, e. g., extreme nervousness, disorders of sleep, stuttering, chorea, incontinence of urine, tetany, and convulsions. The great liability to convulsions depends not only upon the greater irritability of the peripheral nerves, but upon the instability of the nervous centres and the lack of inhibition over the motor ganglion cells of the spinal cord. The nervous centres are more easily exhausted than later in life. Prolonged or continuous overstrain from any cause whatsoever, frequently leads to headache and chorea, and sometimes even to epilepsy and insanity.

Another peculiarity is the serious consequences which often follow reflex irritation, although this is rarely the only factor in the case. Conditions which in adult life produce almost no effect may in infancy be the cause of most alarming symptoms. As a few examples may be cited, reflex symptoms due to phimosis or to intestinal worms, convulsions from disturbances of digestion, nervous symptoms due to eye-strain, or to adenoid growths of the pharynx. In the production of some of these, especially attacks of convulsions, there are several factors, such as the great irritability of the peripheral nerves, the instability of the nervous centres—often a result of disturbed nutrition, as in rickets—and the lack of inhibitory action of the cortex of the brain.

As a third point of importance may be mentioned the grave permanent results which often follow relatively small organic lesions. A good illustration is seen in the lesions which produce cerebral birth-palsy. Here the damage is only in small part the immediate effect of the hæmorrhage, for this often is not great, but it is the interference with the development of certain parts of the cortex that makes this condition so serious.

From what has been said, it follows that the hygiene of the nervous system is of the utmost importance in infancy and childhood. It is essential for the healthy development of the nervous system that all stimulants should be avoided—not only tea, coffee, and alcohol, but undue and unnatural excitement, the effect of which in infancy is almost as serious. A normal development can take place only in the midst of quiet and peaceful surroundings, with plenty of time for rest and sleep. The conditions of modern life, especially in cities, are such that these laws are almost invariably violated, and the consequences of this are seen in the marked and steady increase in nervous diseases among children.

CHAPTER II.

GENERAL AND FUNCTIONAL NERVOUS DISEASES.

CONVULSIONS.

ALL young children, but especially infants, are extremely prone to convulsive disorders, which are manifested clinically in great variety. In certain infants, particularly those who are rachitic, this susceptibility is greatly heightened. To this condition of extreme liability to convulsive attacks the term *spasmophilia* is frequently applied. The convulsive disorders of infancy are: (1) attacks of eclampsia or general convulsions, the type best known; (2) tetany with carpo-pedal spasm; (3) laryngismus stridulus or crowing attacks; (4) the less typical, holding-breath spells, which are apparently a minor form of a general convulsion. Besides these, there are seen in infants a great variety of attacks, which recur from time to time, over quite a long period frequently, of a very doubtful character, until finally they develop into one or other of the types just mentioned. All these convulsive disorders are closely related to one another and an alternation of type from time to time is common. The general etiology of these conditions is still obscure. Their association with rickets is certainly very close. There is also ground for believing that in many of these children there is a disturbed calcium metabolism.

Under the head of convulsions are included attacks of acute transient nervous disturbance, characterised by involuntary rhythmical spasm of the muscles, either of the face, trunk, or extremities, or all of them, usually accompanied by loss of consciousness. They may be regarded as "motor discharges" from the cortex of the brain.

**Etiology.**—The principal predisposing causes are infancy, conditions affecting the nutrition of the brain, and hereditary influences. Of all these factors, the most important one is the instability of the nerve centres which is characteristic of infancy and is associated with the non-development of the voluntary centres of the cortex. The brain grows more during the first year than in all later life, and this rapidity of growth is in itself an important predisposing cause of functional derangement. After infancy, attacks of convulsions are much less frequent, and after seven years they are relatively rare. While convulsions occasionally occur in children previously healthy, the majority of attacks are in those in whom there is at least some disturbance of the nutrition of the brain—the cerebral instability of infancy being greatly exaggerated by such nutritive disorders. The most frequent one is rickets, which may be regarded as altogether the most important predisposing cause of infantile convulsions. They are often one of the earliest symptoms of that dis-

case, and when convulsions occur in infancy without evident cause, rickets should always be looked for. Any disturbance of nutrition, such as is seen in status lymphaticus, syphilis, anæmia, malnutrition, and exhaustion resulting from any acute disease, especially one of the digestive tract, may predispose to convulsions. Children who inherit from their parents a peculiarly nervous temperament are more liable to convulsions than are others. This predisposition is often seen in several members of the same family. The younger the child the greater the susceptibility. Females are rather more frequently affected than males.

The exciting causes include a wide variety of pathological conditions, among which disturbances of digestion take the first place. Where the susceptibility is very great, the exciting cause may be a trivial one. These causes may be grouped under three general heads: (1) direct irritation of the cortex of the brain; (2) reflex irritation; (3) toxic influences.

Under the head of direct irritation may be included all convulsions occurring with the various forms of cerebral disease; the most frequent are meningitis, meningeal or cerebral hæmorrhage, tumour, abscess, hydrocephalus, embolism, and thrombosis. As examples of reflex irritation may be classed the convulsions following severe injuries, renal or intestinal colic, retention of urine, phimosis, or a foreign body in the ear. A case has been reported to me in which the application of cold to the skin repeatedly induced convulsions. Other conditions classed under this head are dentition and worms, but both must be regarded as exceedingly rare causes of convulsions. The exciting cause is very frequently the presence in the stomach or intestines of undigested food; such attacks are sometimes ascribed to reflex irritation, but the majority are better regarded as toxic. Acute and chronic indigestion are to be ranked among the most frequent causes of convulsions, both in infants and older children. In either there may be but one attack, or attacks may recur at intervals of a few months with a repetition of the cause. Of toxic origin may be considered not only the convulsions resulting from conditions like uræmia and asphyxia, but also those which occur at the onset or in the course of various infectious diseases, sometimes classed as febrile convulsions. They are very frequent at the onset of certain diseases, particularly pneumonia, scarlet fever, malaria, acute indigestion, and gastro-enteric intoxication. In these cases the convulsions seem due partly to the intensity of the poison and partly to the suddenness with which it affects the nervous system. Convulsions occurring late in the course of many diseases may be due to toxic influences, especially when associated with exhaustion of the nerve centres, from the prolonged disturbances of nutrition accompanying the febrile condition.

In pertussis, which of all infectious diseases is the one in which convulsions are most frequent, several factors may be present: asphyxia due to a severe paroxysm, cerebral congestion or hæmorrhage resulting

from such a paroxysm, or simply from the peculiar susceptibility of the patient brought about by the disease itself.

Convulsions ending fatally are not infrequently associated with enlargement of the thymus gland. I have seen several such where there was found at autopsy great enlargement of the thymus, which weighed from one to one and a half ounces. Some of these infants were previously healthy; some were rachitic. The similarity of all these cases indicated that the convulsions were in some way due to the enlarged thymus, but the exact explanation is not yet understood.

Frequently recurring convulsions in infancy are very often associated with tetany. The symptoms of the latter condition may be so slight as to be readily overlooked; or there may be no symptoms present except the characteristic electrical reactions.

One attack of convulsions, whatever the cause, renders the patient more liable to a second, and where there have been several, they occur from causes which are less and less marked.

An important element in the convulsions of infancy, according to Hughlings Jackson, is the lack of development of the higher cerebral functions, in consequence of which they do not exert the controlling influence over the discharge of nerve force which they do in later life.

The condition of the brain in the beginning of an attack of convulsions is one of anæmia; this is shortly followed by venous hyperæmia which may be very intense. In infants who die during convulsions the brain and its meninges are usually found intensely congested. They may be the seat of punctate hæmorrhages, and sometimes of more extensive ones. The lungs are also deeply congested, and the right heart is generally distended with dark clots. The other lesions found are accidental.

**Symptoms.**—In some cases prodromal symptoms are present, such as extreme restlessness, irritability, slight twitchings of the muscles of the face, hands, feet, or eyelids. More frequently, however, the attack comes quite suddenly with little warning. Usually the first thing noticed is that the face is pale, the eyes fixed, sometimes rolled up in their orbits; in a moment or two convulsive twitchings begin in the muscles of the eye or face, or in one of the extremities, which usually rapidly extend until all parts of the body participate. In most cases the convulsions become general, but they may remain unilateral even when not due to a local cause—a point which is often forgotten. The contraction of the facial muscles causes a succession of grimaces; the neck is thrown back; the hands are clenched; the thumbs buried in the palms; and a quick spasmodic contraction of the extremities occurs. There may be some frothing at the mouth, and in all true convulsions there is loss of consciousness. Respiration is feeble, shallow, and may be spasmodic. The pulse is weak; it may be slow or rapid; often it is irregular. The

forehead is covered with cold perspiration. The face is first pale, then becomes slightly blue, especially about the lips. Unnatural rattling sounds may be produced in the larynx. The bladder and rectum may be evacuated. The convulsive movements consist in an alternation of flexion and extension occurring rhythmically. All varieties of tonic and clonic spasm may be seen, and in all degrees of severity. The contractions of the two sides of the body are usually synchronous. After a variable time, from a few moments to half an hour, the convulsive movements are gradually less frequent, and finally cease altogether, usually leaving the patient in a condition of stupor. They may recur after a short time or there may be but one attack. A period of general relaxation usually follows the convulsive seizures, frequently accompanied by marked evidences of prostration. Transient paralysis, apparently due to exhaustion of the nerve centres, is not an uncommon sequel.

Death may take place from a single attack; this, however, is rare except in very young infants, especially those who are rachitic or are suffering from status lymphaticus. There may be no sequel to the convulsions if the cause is a temporary one, or they may produce some serious brain lesion, particularly meningeal hæmorrhage. Death from convulsions is generally due to asphyxia, or to exhaustion from the rapidly recurring attacks. Many cases recover in which the children for several minutes had the appearance of being moribund.

One attack of convulsions is very apt to be followed by others; for the occurrence of the first one usually reveals a peculiar susceptibility of the nervous system, and each succeeding attack comes from a less powerful exciting cause than the previous one. The longer the interval which has passed, the less likely is there to be a repetition, especially if the child has passed its third year. The number of attacks may be very great. In one case that I saw, an infant during the latter part of its second year had during six months over thirty-five hundred distinct attacks of convulsions. For a considerable period they reached the almost incredible number of eighty a day, and yet the mental condition of the child in the interval was apparently normal.

**Diagnosis.**—There can rarely be any difficulty in recognising an attack of convulsions. The difficulty consists in determining with which of the many possible exciting causes we have to do in the case before us. If it comes with acute symptoms does it depend upon a cerebral lesion, or does it mark the onset of some other acute disease? Is it reflex, and if so to what is it due? If there are no acute symptoms, is it epilepsy? To answer these questions a careful history must be obtained, and all the circumstances surrounding the patient, the character of the convulsions, and all the other symptoms present must be taken into consideration.

In infancy, epilepsy is the least probable diagnosis. In older chil-

dren the important points indicating that disease are: the presence of some of the stigmata of degeneration, a history of previous attacks, a distinct aura preceding the seizure, or a sudden onset with a cry or fall, biting of the tongue, a tonic spasm preceding the clonic, a deep sleep following the seizure, and, finally, perfect recovery in the course of a few hours. Convulsions which come on with high fever, even though a patient may have repeated attacks, are seldom epileptic. However, in some cases only prolonged observation can enable one to decide positively whether or not epilepsy is present.

Convulsions occurring in brain disease, except acute meningitis, are not as a rule accompanied by any marked rise in temperature. Focal symptoms are often present, such as localised paralysis or rigidity, changes in the pupils, and strabismus. The convulsive movements are frequently limited to one side of the body. It should, however, be borne in mind that unilateral convulsions, even when repeated, do not always mean a local lesion, as I have seen proved by autopsy more than once. In hæmorrhage or meningitis, convulsions are likely soon to recur. In tumour they may recur after a longer interval.

Convulsions may be thought to indicate the onset of some acute disease when they occur in a child over two years old, and when they come on suddenly or with only slight premonition in a child previously well; but the most important point is that they are accompanied by a high temperature— $104^{\circ}$  to  $106^{\circ}$  F. Acute meningitis is the only other condition likely to produce these symptoms. Whether the convulsions mark the onset of lobar pneumonia, scarlet fever, or some other disease, can be determined only by carefully watching the patient's symptoms for twenty-four or thirty-six hours.

In infants, derangements of the digestive tract should first be suspected; in very young infants relatively slight disorders may cause severe and repeated convulsions. In the first weeks of life one may often be in great doubt as to the cause of convulsions. Such attacks may be due to some disorder of the digestive tract, to a recent cerebral lesion like hæmorrhage or to a defective brain development. Sometimes nothing but the progress of the case will definitely clear up the diagnosis.

Examination of the urine should not be omitted in any case of convulsions of doubtful origin. Asphyxia may be suspected in the case of convulsions occurring in the newly born, late in pneumonia, in some cases of pertussis, in spasmodic or membranous laryngitis, or in laryngismus stridulus. Dentition and worms should be considered among the least probable, never as the most probable, causes of reflex irritation, and should not be so accepted without positive evidence. Worms are so rare in infancy that at this period they may be practically ignored. Dentition seldom causes convulsions except in patients who are markedly rachitic. In all cases of convulsions of doubtful or obscure origin oc-

currence in infants, rickets should be suspected as the underlying cause, and the child carefully examined for other evidences of that disease. The close association of convulsions with tetany should not be forgotten.

**Prognosis.**—This depends upon the age of the patient and the cause of the convulsions. Idiopathic or reflex convulsions are rarely dangerous to life except in very young or in rachitic infants. Convulsions associated with enlarged thymus are often fatal. Convulsions occurring at the onset of acute febrile diseases are seldom fatal, and not often serious; they may not even indicate an unusually severe type of the disease. Especially fatal are the convulsions of pertussis and of asphyxia when they occur late in any form of laryngeal or pulmonary disease. In nephritis, while always serious, convulsions are by no means invariably fatal. The conditions during an attack which should lead one to make a bad prognosis are when the convulsions are prolonged or recur frequently; also the presence of very great prostration, a feeble pulse with cyanosis, or deep stupor.

In the prognosis one must take into account not only the immediate result of the attack, but its possible outcome. In a highly nervous or susceptible child a convulsion often means very little. Permanent injury to the brain, simply as a result of an attack, I believe, to be very rare. The possibility of epilepsy is to be borne in mind in all cases where children over two years old have occasional attacks of convulsions, although it is unusual that this result is seen. The farther apart the attacks are and the more definite the exciting cause, the less likely is this to be the case.

**Treatment.**—Summoned to a child in convulsions, a physician should go at once and remain until the attack has subsided. He should take with him chloroform, a hypodermic syringe with morphine, a soft catheter or rectal tube, and a solution of chloral. In order to treat convulsions intelligently one must have in mind the prominent pathological conditions. These are: acute cerebral hyperæmia, a more or less severe asphyxia with pulmonary congestion, an overtaxed right heart, and a tendency to congestion of all the internal organs. The nervous centres are in a condition of such unnatural excitability that the slightest irritation may bring on convulsive movements when they have temporarily subsided. The patient should therefore be kept perfectly quiet, and every unnecessary disturbance avoided. Cold should be applied to the head—best by means of an ice cap or cold cloths—and dry heat and counter-irritation to the surface of the body and extremities. The time-honoured mustard bath causes so much disturbance of the patient that it can usually be dispensed with and the mustard pack substituted. The feet may be placed in mustard water while the child lies in its crib. The mustard pack and footbath should be continued until the skin is well

reddened. The degree to which counter-irritation of the skin should be carried will depend upon the condition of the pulse and the cyanosis.

In controlling convulsions the three remedies which may be depended upon are the inhalation of chloroform, morphine hypodermically, and chloral. Chloroform is undoubtedly the most reliable remedy for an immediate effect, and should be used even in the youngest infant. At the same time that it is being administered, chloral should be given *per rectum*. The initial dose should be, at six months, four grains; at one year, six grains; at two years, eight grains, dissolved in one ounce of warm milk. It should be injected high into the bowel through a catheter, and prevented from escaping by pressing the buttocks together. It may be repeated in an hour if necessary. The effect of the drug is generally obtained in twenty minutes. If, in spite of the chloral, the convulsions show a marked tendency to continue as soon as the chloroform is withdrawn, or if the enema of chloral has been expelled, morphine should be given hypodermically. When the heart's action is weak, this is probably the best of all remedies. Objections are urged against it only by those who have had no experience with its use. To a well-grown child two years old,  $\frac{1}{8}$  grain may be given; one year old,  $\frac{1}{16}$  grain; six months old,  $\frac{1}{32}$  grain. This dose may be repeated in half an hour if no effect is seen. The tolerance of opium in cases of convulsions is very marked, and sometimes double the doses mentioned may be required. The only other agent of much value is oxygen. I have seen convulsions which continued in spite of all other means yield immediately to oxygen. This is most likely to be valuable in cases of convulsions due to asphyxia.

When once under control, the recurrence of the convulsions may be prevented by keeping the patient for two or three days under the influence of chloral with bromide of sodium, the amount of chloral being gradually reduced. If it is badly borne by the stomach and not easily retained by the rectum, either antipyrine or phenacetine may be used with the bromide. Where there is a strong tendency to recurrence of the convulsions, urethan is sometimes even more efficient than chloral. It may be given in the same or in slightly larger doses.

As soon as the convulsions have ceased, the cause should be sought and treated. In infancy it is wise in every case to irrigate the colon thoroughly with warm water, to remove any possible source of irritation. If there is reason to suspect the presence of undigested food in the stomach, this may be washed out. Much more frequently it is in the intestines, and free purgation by calomel is advisable. If there is high temperature, this should be reduced by the cold bath or pack. Secondary attacks are to be prevented by careful feeding, by improving the general nutrition by means of fresh air, iron, cod-liver oil, and phosphorus. The last two are especially valuable in cases due to rickets.



## TETANY.

Tetany is a condition characterised by extreme nervous and muscular irritability with tonic muscular spasm, which may be intermittent or continuous. It usually affects the muscles of the extremities, especially the hands and feet, more rarely the neck, face, and trunk. When limited to the hands and feet it is known as *carpo-pedal spasm* or *arthrogryposis*; and although sometimes classed separately, this is really only one manifestation of the same general condition. In infants, tetany is very frequently associated with laryngismus stridulus, this being present in fully two-thirds of the cases; but in older children this association is quite rare. General convulsions occur in from twenty to thirty per cent of the cases. Although tetany is not a very common disease in America, I believe that it is very often overlooked. In my hospital service I seldom see fewer than a dozen cases a year.

**Etiology.**—While tetany may occur at any age, it is most frequent in infancy. Fully two-thirds of the cases are seen in the first two years of life. It is most common between the fourth and tenth month. Most of the attacks are seen in the winter months. In infancy, males are much more frequently affected. At this age it is rarely seen except when associated with rickets. It may follow broncho-pneumonia, pertussis, typhoid fever, rheumatism, or measles. There is usually present some derangement of the digestive tract. There may be acute diarrhoea or chronic gastric or intestinal indigestion. It is seen in rare cases with intestinal worms and with intussusception. The most common exciting cause appears to be an intoxication from the digestive tract or the irritation of undigested food. Attacks in older children are very uncommon in this country. In girls, tetany may occur at the time of puberty, especially when menstruation is delayed. In animals and in man tetany regularly follows the complete removal of the parathyroid glands. Some pathologists consider the essential cause to be an absence of the secretion of the parathyroid or some disturbance of its function. While this may be accepted as one of the causes of tetany, it is by no means established that it is the only cause. Considerable evidence has accumulated that tetany is in some way associated with disturbances of calcium metabolism; but in what way has not yet been proven. Much regarding the nature and cause of tetany remains to be solved by further investigation.

**Pathology.**—Up to the present time the only constant anatomical lesions demonstrated in tetany are in the parathyroid glands. The most frequent one is hæmorrhage which may be recent, or if old, other changes are present such as the formation of small cysts and pigmentation. While parathyroid changes have been found in many cases they are not uniformly present.

**Symptoms.**—The spasm may develop abruptly, or it may be preceded by sensory disturbances, such as pain, numbness, or tingling. The upper extremities are usually first affected, the spasm gradually becoming more severe and finally involving the lower extremities. Both sides of the body are equally affected. The position assumed by the hands



**FIG. 104.—TETANY, SHOWING THE CHARACTERISTIC POSITION OF THE HANDS AND FEET.**  
In a child two years old.

is very characteristic: The fingers are flexed at the metacarpophalangeal joints and the phalanges extended; the thumbs are adducted almost to the little finger; the wrist is flexed at an acute angle, and the whole hand drawn somewhat to the ulnar side. If the spasm is very marked no motion is allowed at the wrist, but movements at the elbow and

shoulder are usually normal. The feet are strongly extended, sometimes in the position of typical equino-varus. The first phalanges of the toes are flexed, and the second and third rows extended; the plantar surface is strongly arched, and the dorsum of the foot is very prominent, standing out like a cushion. The typical position of the hands and feet is well shown in Fig. 104. The tendo-Achillis stands out prominently. Motion at the hip and knee is generally free. The spasm in many cases is limited to the hands and feet; more rarely the muscles of the thigh, usually the adductors, may be involved. In very rare cases the muscles of the trunk, the face, or the eye may be affected.

The knee-jerk and the cutaneous reflexes are exaggerated, and there is abnormal response to mechanical irritation. Light percussion upon a nerve trunk often induces marked contraction of the muscles supplied by the nerve. This is particularly striking in the face. The contraction of the facial muscles following such irritation is known as "Chvostek's symptom" or the facial phenomenon. A spasm causing the characteristic position of the hands or feet may be excited by pressure upon the nerve trunks, or by constricting the limb so as to cut off the circulation. This is known as "Trousseau's symptom." The most diagnostic feature of tetany is the electrical reaction. It is best obtained in the peroneal nerve. Under normal conditions there may be no contraction to the cathodal closure with a current of less than five milliamperes. In tetany such a contraction is regularly obtained with a current of this strength and often with a much weaker one. Also, a reaction highly suggestive of tetany is an anodal opening contraction with a current of less than five milliamperes, and less than one causing an anodal closure contraction. The most diagnostic reaction, however, is a cathodal opening contraction with a current of less than five milliamperes or a tonic contraction with cathodal closure with less than five milliamperes.

Evidences of pain owing to the spasm are frequently present. It may be so severe as to cause children to cry out. Pain is induced by any attempt to overcome the spasm, and sometimes it is constant. There is no loss of consciousness and no fever. The muscular contraction is generally continuous, although there may be periods of remission or even of intermission. When associated with laryngismus stridulus, the spasm is much increased during these attacks.

The duration of tetany is from a few days to several weeks. The mild form, which is usually seen in infants, in many cases passes away spontaneously in one or two weeks, although there may be relapses and recurrences at variable intervals. The most important complication is general convulsions. These may come on at any time in the course of the attack. Spasm of the glottis may either precede or follow tetany, and by many is regarded as part of the disease. When associated they generally cease at the same time.

**Diagnosis.**—The diagnostic features of tetany are bilateral spasm—in infants usually limited to the hands and feet—without loss of consciousness, the spasm being increased or excited by pressure upon the arteries or nerves, exaggerated reflexes, and the characteristic electrical reaction. Evidences of rickets are usually present. While the other symptoms of tetany are subject to considerable variation, the peculiar electrical reactions are always present and therefore diagnostic. Accepting this reaction as the pathognomonic sign of the disease, it will be found that tetany is often present when not suspected, and that many obscure nervous symptoms are due to this disease which otherwise might be misinterpreted.

The severe form of tetany has been taken for tetanus; but that disease is very rare except in the newly born, and trismus is generally the first symptom. Trismus is extremely rare in tetany. From meningitis and other forms of cerebral disease tetany is distinguished by the absence of cerebral symptoms.

**Prognosis.**—Tetany *per se* is not fatal, but death may result from the development of general convulsions or in infants from the condition, usually some serious disturbance of digestion, which tetany complicates. If recovery occurs it is usually complete.

**Treatment.**—The first indication is to discover and if possible remove the cause, and this in most cases is found in the digestive tract. If rickets is present it should receive the usual treatment, both dietetic and medicinal. For the relief of the spasm, the hot bath is a valuable remedy. This may be repeated two or three times a day. Drugs which have the power of allaying spasm should be given—bromides, chloral, or antipyrine.

The specific treatment of tetany by parathyroid extract has not in my hands been followed by any appreciable benefit. I have seen it tried only in infants. Those who hold the cause to be a disturbance of calcium metabolism, would treat tetany by withholding calcium salts, or by administering them, according to their view of the part which calcium plays in etiology. Whether calcium is given or withheld, seems to me to have no special influence upon the disease. I have seen no advantage in excluding milk and have seen the most satisfactory results when the feeding was carried on according to the indications afforded by the child's digestive symptoms, disregarding the tetany. In prolonged cases there is no doubt that the administration of cod-liver oil and phosphorus is beneficial. They are to be used as in rickets.

#### LARYNGISMUS STRIDULUS—LARYNGO-SPASM.

Laryngismus stridulus is a rather rare condition and belongs especially to infancy. It is most frequently seen in children who are rachitic,

them the term "holding-breath spells" has been applied. They are seen most frequently in the latter part of the first and during the second year, and affect children of the extremely nervous type. Most of them are rachitic. The attacks may occur five or six times a day, or at intervals of several days. Beginning in infancy they may recur from time to time until the age of four or five years. In susceptible children almost any form of excitement may precipitate an attack. By far the most frequent are temper and fright. If anything is attempted to which the child strongly objects, e. g., a cold bath, inspection of the throat, or taking away a toy, an attack may ensue. The child's face becomes flushed, then livid; there is general rigidity of the trunk and extremities, but rarely clonic spasm. This rigidity is followed by complete relaxation with loss of consciousness. The entire attack usually lasts about half a minute. There may be a crowing sound as the child catches his breath or there may be none. After a few minutes of quiet the child gets up and in a short time is apparently as well as ever. Most of those who are subject to attacks of this sort sooner or later have one or more general convulsions. Although in infancy these seizures may recur with alarming frequency, and extend over a period of several years, in most cases with time and with improvement in general health they gradually become less and less frequent until finally they cease altogether. I have not seen these attacks accompanied by tetany, nor followed by epilepsy.

In this condition there is apparently no effort on the part of the child to control his impulses, he simply "lets himself go." Parents, witnessing attacks coming on after correcting or disciplining a child, soon fall into the habit of indulging him in everything with the hope of avoiding them. Such advice, indeed, is often given by physicians. I believe it to be unwise. A much better plan seems to be to teach the child to control himself in everything no matter how small. While it is impossible to assert that the attacks can be brought on at will, such certainly seems at times to be the case, and the development of the will power by every form of self-control seems to exert an influence in preventing these attacks, certainly in children who have reached the age of four or five years.

The treatment of these children is first addressed to the general nutrition; many of them are anæmic and under weight. The feeding and general routine should therefore be the first concern. A life as much as possible in the open air and in the country is most desirable with freedom from every form of nervous excitement or undue nervous stimulation. They should be controlled, taught self-control, and treated tenderly, but with great tact and firmness. Drugs directed specifically to the control of the attacks have in my experience been of little value.

## EPILEPSY.

Epilepsy may be defined as a disease in which there is an established disposition to convulsions of a certain type, with loss of consciousness, which have recurred until a habit of convulsions has become fixed.

A distinction must be made between cases of so-called "idiopathic" epilepsy and those which are secondary to a definite lesion of the brain, such as tumour, sclerosis, or abscess. Convulsions of the latter character are designated as "symptomatic" epilepsy, and are discussed in connection with the various diseases in which they occur. The nature of the attack may, however, be identical in both varieties, and may not differ from an ordinary attack of convulsions or eclampsia.

The proportion of idiopathic cases in children is not so large as was formerly supposed; many of these have been shown to depend upon lesions once overlooked, particularly mild infantile cerebral paralyses.

**Etiology.**—From a consideration of 1,450 cases of epilepsy, Gowers states that twelve per cent begin in the first three years of life, and forty-six per cent between ten and twenty years. The greatest tendency to the development of the disease is shown about the time of puberty. Females are rather more liable to be affected than males, although the difference in sex is slight. Heredity plays an important rôle in the production of the disease. In one-third of the cases, according to Gowers, there is a family history either of epilepsy or insanity.

Not very infrequently epilepsy may be traced to convulsions occurring during infancy. Infantile convulsions are very common, and usually the cause which produces them is a transient one. The proportion of such cases which develop epilepsy later in life is certainly very small. One frequently meets with children from two to five years old who have occasional attacks of convulsions, often from apparently trivial causes. In my experience, the great majority of these also recover completely with proper treatment; a very few become epileptic. The first seizure is sometimes traceable to fright, great excitement, heat-stroke, or blows or falls upon the head even without any gross lesion. As reflex causes may be mentioned intestinal worms, phimosis, adenoid vegetations of the pharynx, delayed or difficult menstruation, and masturbation. Most of these are rare causes, but they may be sufficient to produce the disease where a strong predisposition exists.

Among the most important factors in producing a paroxysm, is intestinal putrefaction associated with chronic constipation and chronic intestinal indigestion. I believe it to be one of the most important etiological factors in cases occurring in children, particularly as an exciting cause of the first attacks.

**Pathology.**—It is not within the scope of this work to discuss the various theories which have been advanced. The following are the conclusions reached by Gowers:

“The muscular spasm is to be regarded as the result of the sudden overaction (discharge) of nerve cells, the violent liberation of nerve force, and the sensations which the patient experiences before losing consciousness must be due directly or indirectly to the same cause. The disease which excites convulsions is most frequently at the cortex, and when organic disease causes convulsions that begin locally, the disease is almost invariably at the cortex. In idiopathic epilepsy the convulsions sometimes begin in this way, and this suggests very strongly that in such cases the change occurs in the cortex. Epilepsy must then be regarded as a disease of the gray matter, most frequently of the gray matter of the cortex.”

While there is pretty general agreement that the seat of the morbid changes in true epilepsy is in the cortex, but little is yet definitely known as to the nature of these changes. It is probable that a great variety of lesions, many of which are apparently slight, may produce this disease.

**Symptoms.**—Two distinct types of epileptic seizures are met with: the major attacks, or *grand mal*, in which there are severe convulsions lasting from two to ten minutes, with loss of consciousness, etc.; and minor attacks, or *petit mal*, in which the convulsive movements are slight and may be absent, and in which the loss of consciousness is often but momentary. Between these two extremes all gradations are seen.

*Grand Mal.*—The onset may be sudden, without premonition, or it may be preceded by certain prodromal symptoms known as the aura. The aura may be motor, such as a local spasm of the hand, face, or leg; or sensory, such as numbness and tingling in any part of the body, or some abnormal sensation rising gradually to the head, at which time loss of consciousness occurs. The variety of sensations described by patients as indicating an attack is endless. There may be a sensation in one finger, in the face, tongue, eye, or in any part of the body; or the warning may be of a general character, like a tremor or a shivering sensation, or a feeling of faintness. There has also been described a visceral or pneumogastric aura, in which there is epigastric pain, sometimes nausea, and a sensation of a ball in the throat; or there may be palpitation, or cardiac distress. There may be general giddiness or vertigo, or a sensation of fulness in the head; or feelings of strangeness, or a dreamy, dazed condition; and, finally, the aura may have reference to any of the special senses, most frequently to sight. Sparks may appear before the eyes, or flashes of light or colour, or strange objects may be seen; or there may be a momentary loss of hearing; or strange sounds may be heard. In most cases the aura is peculiar to the individual.

At the beginning of the seizure the face becomes pale, the pupils

widely dilated, the eyes rolled up in their orbits and fixed. Speedily there is loss of consciousness. Simultaneously with these symptoms, or immediately following them, there occurs a violent tonic muscular spasm to which are due the characteristic symptoms of the early part of the seizure, viz., the fall, cry, biting of the tongue, cyanosis, and evacuation of the bladder or rectum. The fall is forcible, violent; in fact, the patient is precipitated usually forward, and frequently suffers injury, never sinking down as in a faint. The head is often strongly rotated to one side. The position of the hands is frequently that assumed in tetany. The cry is a hoarse, inarticulate sound, not very loud, and is due to forcible expiration, owing to spasm of the muscles of respiration with the glottis partially closed. The cyanosis is the result of tonic spasm of the muscles of respiration; it may be quite intense, so that the face is livid, bloated, and the features distorted. The spasm of the muscles of mastication causes the biting of the tongue. Evacuation of the bladder and rectum may result from contraction of their walls, or from spasm of the abdominal muscles. The violence of the muscular spasm in this stage may be very great; it has caused fracture of bones, rupture of muscles, and even dislocation of joints.

The stage of tonic spasm may be only momentary, the patient passing almost at once into the stage of clonic convulsions. The usual duration is from ten seconds to half a minute. In the stage of clonic spasm which follows, the symptoms are those of an ordinary attack of convulsions. The muscular contractions are violent, and there is often frothing at the mouth. Gradually the muscles of respiration relax, air enters the lungs, and the cyanosis passes off. After the clonic spasm has continued for a variable time—from two or three minutes to half an hour—the muscular contractions become less and less frequent, and finally cease altogether. In a few minutes the patient may regain consciousness, look vacantly around, and in a dazed way perhaps ask what has happened, he being completely oblivious to all that has occurred. More frequently, however, he passes at once into a deep sleep, which continues for an hour or more, but from which he can be aroused. From this he usually awakens with a severe headache, which may continue for several hours. After this he often feels better than for several days preceding the attack. During the seizure the temperature may be elevated one or two degrees, but rarely more. The attack may be followed by a slight temporary paresis, or aphasia, hysterical phenomena, vomiting, and intense hunger. In very rare cases the urine may contain a trace of sugar.

*Petit Mal.*—The minor attacks of epilepsy may present a very great variety of symptoms, and at times it is almost impossible to decide that these are epileptic, except from their periodical occurrence. They pass under the names of “spells,” “attacks of dizziness,” “fainting turns,”



etc. The most striking thing which stamps them as epileptic is the loss of consciousness, and this may be of short duration, sometimes only momentary, and so pass unnoticed. In some cases it is absent altogether. There is no fall, but there may be a slight dropping of the head, a fixed stare for a moment or two, and that is all. This may or may not be preceded by an aura. After such a mild attack the patient's mind may be somewhat confused, and he may do or say strange things. All sorts of curious acts have been performed in an automatic way by patients in the condition which follows an attack of epilepsy, which may perhaps be regarded as part of the attack. In rare instances even acts of violence may be done.

*The Mental Condition of Epileptics.*—A careful distinction should be made between cases in which epilepsy is secondary to some organic brain disease, and the mental disturbances seen in cases of idiopathic epilepsy. The children who are the subjects of the latter disease, and who are perfectly normal mentally, are certainly few. All degrees of disturbance may be seen, from those who are simply dull, apathetic, backward in development, and uncontrollable in temper, to those who are melancholic, idiotic, and even maniacal. The earlier in childhood epilepsy develops, the greater is usually the mental disturbance seen, because of the effect of the seizures upon the brain during its period of active growth.

*Symptomatic Epilepsy.*—This occurs most frequently in children as a sequel of cerebral palsy, usually with hemiplegia, and it may follow either the congenital or acquired form. Epilepsy may come on at any time after the onset of the paralysis—from a few months to five or six years. At first the attacks may be separated by long intervals, but they gradually become more frequent as time passes. The convulsions in post-hemiplegic epilepsy begin, as a rule, on the paralysed side, and for a long time they may be confined to that side; but later they may become general, in which case they are indistinguishable from attacks of idiopathic epilepsy. Severe seizures are more likely to be seen than are the mild ones.

*Course of the Disease.*—In most cases seizures at first occur at long intervals, of perhaps a year, but later they become more and more frequent. Either the mild or the severe attacks may be first seen, and may remain throughout as the only type present, or they may be associated in the same case. There are most frequently seen occasional major attacks with a large number of minor ones. The interval between the epileptic seizures in most cases is from two to four weeks, although they may be of daily occurrence. Sometimes three or four seizures will follow one another closely, and then there will occur a long interval of immunity. The seizures may come on either during sleep or in the waking hours, and in some cases for a long time they may occur only in sleep.

Such cases present peculiar difficulties in diagnosis, and are often long unrecognised as epileptic. The general health of patients may be quite normal.

Death rarely, if ever, results from epilepsy, except from some accident at the time of the seizures, or from the condition known as the *status epilepticus*; in this the attacks come on with great frequency and severity, the patient at times passing rapidly from one convulsion into another, the temperature rising to 105° or 106° F., and death occurring either from exhaustion or in coma.

**Diagnosis.**—In most cases there is little difficulty in recognising the major attacks when they occur by day. Nocturnal attacks may be diagnosed by the cry, the biting of the tongue, blood upon the pillow, sub-conjunctival extravasation, evacuation of the bladder or rectum, and the severe headache. Minor attacks present the greatest difficulties, and a positive diagnosis is often impossible until the patient has been watched for a long time. The most important points to be noted are sudden pallor, dilatation of the pupils, temporary loss of consciousness, or simply mental confusion, and sometimes the evacuation of the bladder.

It is not always possible to distinguish between secondary or symptomatic epilepsy and the idiopathic or hereditary form, particularly if the case comes under observation late in the course of the disease. The points which go to establish the first form are: that the convulsive movements are partial, or limited to one side; that when they are general, they always begin in the same part of the body; or that there is a history of partial or unilateral attacks for some time before the occurrence of any general convulsions. It is important in all cases to examine the patient carefully for signs of an old hemiplegia, the symptoms of which may be so slight as to be readily overlooked. A marked increase in the reflexes of one side is quite as conclusive evidence as is a distinct weakness of the arm or leg. In idiopathic epilepsy some of the stigmata of degeneration are usually present. The sudden development of epileptiform seizures in a child previously healthy, and in whom there is no hereditary history of the disease, should always arouse the suspicion of organic brain diseases, especially tumour.

**Prognosis.**—The danger to life in epilepsy is very slight. Death is generally due to some accident, particularly drowning, at the time of a seizure. The tendency to spontaneous cessation of the attacks is small, while the tendency to recurrence is very great.

The prognosis in any given case depends upon the cause of the disease and the duration of the symptoms. When the cause can be removed, and when the symptoms have lasted less than a year, the prospects of permanent cure are fairly good. This is particularly true of cases in which the epilepsy clearly depends upon gross errors in diet, with chronic intestinal indigestion. If an hereditary tendency to the

disease is marked, if the epileptic seizures have developed apart from any adequate exciting cause, and if they have continued untreated or in spite of treatment for two or three years, the symptoms may perhaps be relieved, but there is little prospect of permanent cure. In the cases also which are due to local irritation, like that resulting from an old meningeal hæmorrhage, the prognosis is invariably bad, and only temporary relief is to be expected. A few cases of traumatic epilepsy have been cured and many have been greatly improved by a surgical operation.

**Treatment.**—The first indication is to remove the exciting cause where one can be found. Particular attention should be given to the digestive organs. The most hopeful cases are those associated with disturbances of digestion, especially chronic intestinal indigestion with constipation. These cases are to be managed like others of the same sort in which epileptic attacks are not present. Meat should be allowed once a day and in moderate quantity. Milk should be given, diluted if necessary, also buttermilk and kumyss. Green vegetables, peas and beans, may be given freely; also all fresh fruits. Tea, coffee, and alcohol in every form must be absolutely prohibited. The most careful attention should be given to the bowels. Under no circumstances should a condition of chronic constipation be neglected. A dose of calomel once a week and intestinal irrigation two or three times a week are of great value in many cases. When the symptoms of intestinal putrefaction are marked, borax is at times of value—two grains three times a day to a child of five years—or salicylate of sodium, salol, or the benzoate of sodium may be given; the dose of each being from two to ten grains, according to the age of the child, after each meal. The general hygiene of the patient must receive careful attention. He should lead a simple, regular life, as much as possible out of doors, away from all sources of excitement.

All the foregoing means of treatment are of equal importance with the use of special drugs. The most common mistake is to rely only upon drugs, ignoring the other measures mentioned. It not infrequently happens that drugs are without any effect when they are the only means of treatment employed, whereas in conjunction with other measures marked improvement is seen.

The bromides are unquestionably the best means of combating the epileptic habit. Either the sodium salt alone or a combination of the sodium and ammonium or strontium bromide is to be preferred. The purpose should be to give the smallest doses which will control the seizures. Children require proportionately larger doses than adults, and in most cases a child of five years will need from twenty-five to fifty grains a day. The method of administering the bromides is of some importance. The larger part of the quantity for twenty-four hours should be given shortly before the time when the seizures have usually

occurred; in the interval much smaller doses. In most cases it is desirable to give a full dose at bedtime. Bromides should always be given largely diluted—in from six to eight ounces of water.

Cases of *petit mal* are especially difficult to control. For such there is often an advantage in combining belladonna with the bromides. In all cases the treatment must be continued for a long time if anything is accomplished. The bromides should be gradually reduced after the attacks are controlled, but must be given in moderately large doses for at least two years after the seizures have ceased. Sometimes the combination of chloral or antipyrine with bromides is advantageous, particularly if the latter are badly borne or cause an annoying amount of acne. Seguin states that he has been able to control the acne in many cases by giving at the same time moderate doses of arsenic.

Cases have been reported of very striking benefit following the use of calcium lactate. It should be given in full doses, at least thirty grains a day for a considerable period.

The surgical treatment of epilepsy has of late attracted much attention. An operation is to be considered in cases in which the paroxysms are very frequent and severe, and when there is present a definite local cause, such as an old fracture of the skull, or when epilepsy has followed an injury to the head even without fracture.

The education of epileptic children is a subject of great difficulty and is often neglected. There are many reasons why it is impracticable to send them to ordinary schools, and it is therefore very desirable that special schools and colonies for them should be established.

*The Management of the Attack.*—Abortive measures are sometimes successful in cases with a distinct aura, the most reliable being the inhalation of nitrite of amyl. While the seizure lasts, the patient should be prevented from injuring himself. The clothing should be loosened, a spool or cork should be placed between his teeth to protect the tongue, but no effort made to restrain his movements unless he is likely to do violence to himself. An epileptic child should never be without some companion.

## CHOREA.

(*Saint Vitus's Dance.*)

Chorea is a functional nervous disease characterised by aimless, irregular movements of any or all the voluntary muscles. Choreic movements are of a somewhat spasmodic character, often accompanied by an apparent or real loss of power in the groups of muscles affected, and by a mental condition of extreme irritability.

**Etiology.**—Chorea is most frequently seen between the ages of seven and fourteen years. Of 146 cases, 6 were under five years, 72 between five and nine years, and 68 between ten and fourteen years. The

youngest case of which I have record was that of a child four years old. It is extremely rare before the third year, although it may occur even in infancy. My own observations coincide with those of nearly all writers, that the disease is more than twice as frequent in females as in males. While chorea may be seen at all seasons, it is much more frequent in the spring months. Of 717 attacks studied by Lewis (Philadelphia), the largest number began in March, and the next largest number in May; in my own cases May stood first.

The relation of chorea to rheumatism is of much importance. The investigations of different writers have given results which are somewhat contradictory. Some have found evidences of rheumatism in but a small proportion of the cases—in not more than five or ten per cent—while the statistics of others have placed the percentage with rheumatism as high as fifty or even sixty per cent. The question hinges largely upon what is to be admitted as evidence of rheumatism in a child; if cases of acute articular inflammation only, then the number will be very small; if subacute cases with joint swellings are included, the proportion will be considerably larger; while if we admit cases of acute endocarditis without articular symptoms, and those of articular pains and joint stiffness but without swelling, the proportion will be very much increased. My own belief is that there is a very close connection between chorea and the rheumatic diathesis as manifested by all the symptoms above noted, and accompanied by a family history of rheumatism. There seems to be a large group of cases, therefore, which may be classed distinctly as rheumatic. There are, however, a few others in which no such element can be found.

My former associate, Dr. F. M. Crandall, has analysed 146 cases of chorea treated by us in an out-patient clinic and in private practice, with the following results: Of 111 cases in which the question of rheumatism was investigated there was a definite history of it in 63. In 41, rheumatism occurred before the chorea; in 13, the first evidence of rheumatism was coincident with the chorea; and in 9 it first occurred subsequently to the chorea, usually within three months. In about one-third of the cases, attacks of rheumatism occurred during or subsequent to the chorea as well as before it. It may then be stated that previous rheumatism was evident in 37 per cent, concurrent rheumatism in 24 per cent, and subsequent rheumatism in 15 per cent of the cases. Excluding cases mentioned twice, and also all those in which there was a history only of "growing pains," there was evidence of articular rheumatism in 56.7 per cent of the cases. Many of these patients have now been under observation for several years, and it has been interesting to see, as time has passed, how the evidences of rheumatism have multiplied the longer the cases have been followed.

In the above statistics only articular symptoms have been accepted as evidence of rheumatism. If the cases of endocarditis without articular

symptoms were included, as I think they might fairly be, it would raise the proportion of rheumatic cases still higher. The great proportion of cardiac murmurs persisting after chorea, if not all of them, should, I believe, be classed as rheumatic, even if no articular symptoms have been present.

Overpressure in school is often an important element in the production of chorea. Anæmia, if not an essential factor, is certainly a very important one, and the great proportion of cases present very distinct evidences of it. Chorea may develop as a sequel of any of the infectious diseases, more particularly scarlet and typhoid fevers. Among the reflex causes may be mentioned phimosis, either lumbricoids or pinworms, delayed menstruation, and ocular defects, although the latter more frequently cause a local spasm of the muscles of the eyes, which can hardly be considered choreic. Hereditary influence is of considerable importance in the production of chorea. It is much more frequent in children of neurotic families, and very often several successive generations, or several children in the same family, may suffer from the disease.

The exciting cause of chorea in a certain proportion of cases is fright; occasionally it arises from imitation, and the disease has been known to occur epidemically in institutions.

The rôle of bacteria in the production of rheumatic chorea is still undecided. The organism which Poynton and Paine have described as the cause of acute articular rheumatism has been found in the meninges of the brain in a few fatal cases of chorea.

**Pathology.**—The exact pathology of chorea is at the present time not settled. The seat of the morbid process is undoubtedly the central nervous system, probably the motor areas of the cortex. The cases associated with rheumatism are now generally regarded as of infectious origin. In some severe cases which were fatal, owing to association with acute endocarditis, capillary emboli have been found in the brain. However, it is by no means established that this is the condition present in most of the rheumatic cases. The fact that in the great majority of such cases complete recovery occurs in the course of a few weeks or months, speaks strongly against any important structural change in the nervous centres. In cases not rheumatic, the most probable explanation of the symptoms is to be found in vascular changes, having their origin in disturbances of nutrition.

**Symptoms.**—An attack of chorea generally comes on gradually. At first the child may be considered simply as unusually nervous; if at school, there may be noticed a difficulty in writing, drawing, or in using the hands for other delicate operations. At home, the child is continually dropping things, has difficulty in feeding himself, sometimes in buttoning his clothes, and very frequently he is not brought to the physician until the symptoms have lasted a week or two. Sometimes

the legs are first affected, and a history is given of frequent falls, a stumbling gait, difficulty in going upstairs, etc. At other times the spasm is first seen in the facial muscles, with disturbance of articulation, twitchings of the eye muscles, and the child may be punished for making grimaces. In most cases the spasmodic movements soon extend to all parts of the body. They remain limited to one side of the body (hemi-chorea) in about one-third of the cases. When fully developed, the movements of chorea are quite unmistakable. They are irregular, jerking, spasmodic, never rhythmical, rarely symmetrical, and vary in intensity from an occasional muscular contraction to almost constant motion. The movements are not under the control of the patient's will, and are usually intensified by efforts to repress them. They are increased by excitement, embarrassment, or fatigue, but do not continue during sleep.

Very often there is weakness of the affected muscles, which may be so great as to lead to the suspicion that actual paralysis exists. Not infrequently I have had patients brought to the clinic for supposed paralysis, either of one extremity or of one side of the body, where the choreic movements have not been severe enough to attract the attention of the mother. This paralysis usually disappears in the course of a few weeks.

In severe forms of chorea the patient may be unable to walk, to speak intelligibly or even to sit up in bed. Control of the bladder or rectum may also be lost. The symptoms may be so intense as even to endanger life. Such cases, however, are dangerous, not from the choreic movements, but from the acute endocarditis with which they are frequently associated.

The mental condition of choreic patients is one of marked irritability. They are fretful, emotional, easily provoked to tears or laughter, and difficult to control. In extreme cases a mental disturbance bordering upon acute mania has been observed. In other cases the facial expression and manner of speech strongly suggest beginning imbecility. All degrees of speech disturbances are seen from the slight difficulty in articulation due to inability properly to control the movements of the tongue and lips, to a condition in which speech is almost impossible. In severe cases speech may be temporarily lost.

Cardiac murmurs are frequent in chorea. Some of these are of anæmic origin, some possibly are due to chorea of the cardiac muscle itself—although this is a matter of some uncertainty—but a large number, probably the majority, are due to concurrent endocarditis, as is shown by the fact that they are permanent, and are followed by all the signs of organic heart disease. During every attack the heart should be closely watched, especially in children in whom there is a strong predisposition to rheumatism.

The general condition of choreic patients is usually much below

normal. They are anæmic; the appetite is poor, often capricious; they sleep very badly; they suffer frequently from headaches; they are easily fatigued by slight muscular exertion; and in short they have all the symptoms of a greatly disturbed nutrition.

**Course and Duration.**—The ordinary form of chorea tends to spontaneous recovery in from six to ten weeks. Exceptionally it may last for three or four months. In a small number of cases the disease may become chronic and continue indefinitely. Certain forms of local spasm, particularly choreiform movements of the muscles of the face, eyes, or neck, may be permanent. In any case of chorea which lasts longer than the usual time, the patient should be carefully examined for some cause of peripheral irritation. The tendency to relapses and second attacks is very marked. Later attacks are likely to occur in the spring succeeding the first illness, and in a small number of patients attacks may come every year for four or five years.

**Diagnosis.**—There is little difficulty in recognising chorea from the sudden, irregular, spasmodic contraction of the muscles coming on under the circumstances indicated. No other movements of childhood are likely to be confounded with it. The form of chorea following hemiplegia is usually more athetoid than choreic, yet at times it closely simulates ordinary chorea. The difficulty in distinguishing between the two is often increased by the fact that the weakness of simple chorea may, if unilateral, closely simulate hemiplegia. The existence of rigidity, contractions, and increased reflexes belongs exclusively to hemiplegic cases, and these will usually suffice to clear up all doubt with reference to the diagnosis.

**Prognosis.**—As a rule, this is favourable, and complete recovery can usually be predicted, the exceptions being few in number. Parents should always be warned of the tendency of the disease to return in succeeding years, and the fact should be stated that in a certain proportion of cases the disease may be permanent. The prognosis of the cardiac murmurs occurring in chorea should always be guarded, although some of these are functional and disappear with recovery from the chorea; but the number of those which do not disappear is sufficiently large to make one always apprehensive as to the ultimate result. Acute chorea accompanied with endocarditis may be fatal; a number of such cases are on record in which there was no other evidence of rheumatism.

**Treatment.**—The general management of the case is equally important with the administration of drugs. A child with chorea should at once be taken from school, and should never be subjected to punishment or to ridicule on account of the movements. Special attention should be given to the patient's diet and general nutrition. Tonics, especially iron, are indicated in most cases. The food should be simple and nutritious, and all stimulants, particularly tea and coffee, should be absolutely



prohibited. While fresh air is desirable, exercise should be prescribed with great caution and its effect should be carefully watched. A certain amount of moral restraint is indispensable; thus it often happens that choreic patients do very badly at home where they are indulged and receive sympathy, while in a hospital, where they are under restraint and made to control themselves, they begin to improve immediately. In all severe cases the "rest treatment" should be employed. It is equally beneficial in the milder ones; the patient is put to bed, and complete mental and physical rest secured. This may be combined with gentle massage for fifteen or twenty minutes a day. The daily use of warm baths, either alone or in conjunction with massage, is decidedly beneficial. In other cases the regular use of cold douches is of value.

With reference to the use of drugs, it is advisable to separate from other cases those in which the connection with rheumatism is very close. In the rheumatic cases, salicylate of soda is often efficient, while the drugs usually employed may be absolutely without effect. In the non-rheumatic cases, arsenic is undoubtedly a valuable remedy. Beginning with four drops of Fowler's solution three times a day for a child of eight years, the daily quantity may be increased by one drop every two or three days until eight drops are given at each dose. One should stop short of this if digestion is disturbed, or there is puffiness of the face or albumin in the urine. Arsenic should always be given after meals, and largely diluted. The possibility of arsenical poisoning should be remembered, although it is rare. Semple has reported a case in which multiple neuritis and general pigmentation of the skin occurred after four weeks' administration of the drug.

Antipyrine and strychnine sometimes succeed where arsenic fails. From fifteen to twenty grains of antipyrine should be given daily in divided doses to a child of eight years. To a child of eight years strychnine should be given in doses of  $\frac{1}{16}$  of a grain three times a day, the dose being gradually increased until double this quantity is given. Acute chorea of great severity may require opium, or bromides and chloral.

In estimating the value of drugs in the treatment of chorea, the natural course of the disease should be kept in mind, since those drugs which are taken after the third or fourth week are much more likely to be thought beneficial than those used in the early period of the attack.

Chorea has a strong tendency to recur, especially in the spring months. Children who have had one attack should be closely watched, particularly with reference to their work in school. They should not be crowded in their studies, they should have long vacations, and the nervous system should not be put upon any severe tension for a long time.

## OTHER SPASMODIC AFFECTIONS.

**Habit Spasm.**—This term is used to describe certain spasmodic muscular movements which at first are only occasionally noticed, but which may persist until they become habitual and almost entirely involuntary. The movements usually affect the muscles of the face, but they may be seen in almost any part of the body. The most frequent varieties consist of blinking or sudden frowning, raising the eyebrows, or some peculiar grimace. At other times there is sudden twisting of the head, shrugging of the shoulders, or jerking of the hands. It is not often seen in the lower extremities, but the muscles of respiration are quite frequently affected. There may be a half-sigh, a sort of sob, or a peculiar dry, laryngeal cough.

These movements are at first infrequent; but as the habit becomes more firmly fixed the spasm recurs every few minutes, and in severe cases it may be almost continuous. The form of spasm is not always the same; one may disappear and another take its place. The condition may last for months or years, and it may even be permanent.

Habit spasm is really little more than exaggerated nervousness continuing in some definite form until by repetition a fixed habit is established. It is different in cause, course, prognosis, and treatment from chorea, with which, however, it is often confounded.

The causes are those of neuroses in general. In the beginning, at least, the general health is usually below the normal. The patients are nervous children of neurotic antecedents. There may be a history of some definite exciting cause, such as illness or overwork in school. There may be some local cause of which the spasm is merely a reflex. Common ones affecting the facial muscles are visual defects, adenoids, and carious teeth.

Habit spasm is to be differentiated from chorea; this is usually easy, from the limitation of the movements to one part or group of muscles and from the duration of the disease.

Treatment is quite unsatisfactory after the habit has become fixed, hence it is of very great importance that it should be arrested at the earliest possible age. Punishments are of no avail, and usually aggravate the condition. Rewards are much more effectual. The general health should receive attention and nerve tonics should be given, especially strychnine.

**Athetosis and Athetoid Movements.**—These terms, introduced by Hammond, are used to describe a chronic form of spasm usually seen in the hand, but sometimes also in the foot, and even the face. It may affect both sides, but in most cases it is unilateral. The movement is slow, irregular, and incoordinate—a sort of “mobile spasm,” it has been called—and there may be associated a certain amount of muscular rigid-

ity. Such movements rarely occur in persons apparently healthy, but are usually seen as a sequel of cerebral palsies, generally hemiplegia. Recovery from the paralysis may be so nearly complete that the athetoid movements are looked upon as primary. In some cases the movements are more rapid and somewhat resemble those of chorea, the condition being sometimes classed as *post-hemiplegic chorea*. Athetosis is not influenced by treatment.

**Rotary and Nodding Spasm of the Head.**—These are rare forms of irregular movements usually observed in infancy. The condition was described long ago by Henoch. The most frequent is the rotary spasm, which consists in a side-to-side oscillation of the head, which may be slow or rapid, and in some cases is almost continuous. Some children have at times the nodding spasm also, and in others this is the only movement seen. Nystagmus is frequently associated, and may affect one or both eyes. In a few of the reported cases convergent strabismus was present.

The causes of the condition are extremely obscure. It is usually seen in infancy between the third and eighteenth months, and, like most nervous symptoms of this period, has been ascribed to dentition, but without any special reason. In three of the cases reported by Hadden, it followed an injury to the head, and might perhaps be regarded as a result of cerebral concussion.

As a rule, the condition lasts for several months and improves, recovery generally taking place. The prognosis is therefore usually favourable.

**Nystagmus.**—This term is applied to rhythmical, involuntary, oscillatory movements usually of both eyes. They are caused by the alternate contraction of opposing muscles. Nystagmus may be either vertical or horizontal. It is most often seen in infants a few months old, and is a symptom of irritation which may be general or local. In some cases the movement is almost continuous, occurring even in sleep; in others, it is only noticed at times of special excitement.

The etiology of nystagmus is obscure, and it may occur in quite a variety of conditions—sometimes referable to the eye, at other times to the central nervous system. On the part of the eye, nystagmus may be due to blindness from any cause, to congenital cataract, corneal opacity, disease of the choroid or retina, or to errors of refraction. It may be seen in almost any organic disease of the nervous system, both with focal and diffuse lesions, especially in chronic hydrocephalus, insular sclerosis, tuberculous meningitis, and in diseases in which sight is impaired. Nystagmus may be of reflex origin, as in a case recently occurring in the Babies' Hospital, where an infant with a severe diarrhoea had repeated attacks, which disappeared each time after intestinal irrigation. While it is of no importance as a localising symptom, nystagmus usually indicates

something more than functional disturbance. An exception to this may perhaps be made when it follows cerebral concussion. In such cases it is usually temporary, disappearing in a few days or weeks. Under most other conditions it may continue indefinitely.

The condition of the eyes should be investigated in every case of nystagmus; it is only when the cause is here, and can be removed, that habitual nystagmus is amenable to treatment.

**Hiccough (Singultus).**—This is a spasm of the diaphragm which is usually seen in young infants. In them it is in most cases due to some irritation in the stomach. It is seen after eating, and may depend upon overfilling of the stomach with food, swallowing of air, etc. In other cases it has no relation to the taking of food, and is to be regarded as a form of reflex spasm, which may occur from a variety of causes, such as cold feet, chilling of the surface during the bath, or suddenly taking an infant from a warm bed into a cold room. In cases like the above, hiccough, though sometimes annoying, is of little importance. It may be associated with gastric indigestion, with intestinal flatulence or inflammation, with peritonitis or intestinal obstruction. With the last two conditions it is always an unfavourable symptom. In older children hiccough sometimes occurs as a pure neurosis.

The object of treatment is to remove the cause. In infants this is to aid in the expulsion of the gas from the stomach by manipulation, by position, or the other means useful in gastric colic. When it is a nervous symptom only, it may be arrested by holding the breath, by prolonged forced expiration, as in blowing a trumpet, and sometimes it may be relieved by drugs which control muscular spasm, e. g., antipyrine or chloral.

**Thomsen's Disease (Congenital Myotonia).**—This rare disease is usually congenital. It may occur in several members of the same family, and is often hereditary. The characteristic symptoms are a peculiar rigidity of the muscles which is observed when they are first brought into action after repose. This rigidity is spasmodic, and usually continues but a few moments. It may recur when voluntary movements are again attempted. If, however, muscular effort is persisted in, it soon passes off. It is increased by apprehension, excitement, or cold, and by observation. The legs are most frequently affected, the condition being often noticed when the patient starts to walk; any of the voluntary muscles, however, may be involved. It may be greater upon one side of the body than upon the other. The muscles are abnormally sensitive to mechanical stimulation, and often to galvanism. They are above normal size, and the fibres themselves are enlarged.

The pathology of this disease is, according to Gowers, an altered functional condition of the muscle fibres, and an abnormal functional state of the nerve cells of the cord and the cortex. It is incurable, although the symptoms may be improved by active muscular exercise.

**Cervical Opisthotonus.**—This is usually a symptom of disease at the base of the brain, occurring with cerebro-spinal, tuberculous, and chronic basilar meningitis, sometimes with tumours of the posterior fossa of the skull. However, in certain cases it occurs as a form of reflex spasm, particularly in young infants who are suffering from diarrhoeal diseases or marasmus. In these cases it may last for days or weeks. The deformity is produced by a contraction of the superior fibres of the trapezius and by the posterior group of cervical muscles.

**Torticollis—Wry-neck.**—Torticollis is usually produced by a tonic spasm of one sterno-mastoid muscle, with which may be associated spasm of the posterior cervical muscles, including the trapezius. In recent cases there is simply a condition of muscular spasm; in those of long standing there may be permanent shortening of the affected muscle, atrophy, and partial paralysis. A somewhat similar deformity may be caused by cicatricial contraction of the tissues of the neck following burns.

The deformity varies somewhat according as the sterno-mastoid muscle is alone affected, or the posterior muscles also, and as to which



FIG. 105.—SPASMODIC TORTICOLLIS. Trapezius and sterno-mastoid of the left side are affected.

predominates. In simple sterno-mastoid spasm the head is inclined to the affected side and rotated toward the opposite side; the chin is raised, and the ear approaches the clavicle. When other muscles are involved the deformity is modified. If the trapezius is affected (Fig. 105) there is less rotation of the head, but it is drawn to the affected side and somewhat backward, while the shoulder is raised and the spine curved. Both of these symptoms may be seen to a slight degree in almost any marked case of sterno-mastoid spasm. Sometimes the spasm of the posterior muscles affects both sides; the head is then drawn

backward and held rigidly but without rotation. In most of the recent cases the deformity can be partially or entirely overcome by passive force; but after a time this is impossible, owing to muscular shortening. In recent cases localised pain and tenderness are also frequently present, and sometimes they are severe.

*Etiology.*—Spasmodic torticollis may be produced by anything causing irritation of the trunk or the branches of the spinal accessory nerve; the source may be in the spinal canal, in the cranium, along the course of the nerve trunk, or of any of its peripheral fibres.

Torticollis may be congenital or acquired. Regarding the cause of congenital torticollis there is some dispute. Such cases have often been attributed to the contraction resulting from hæmatoma of the sterno-mastoid. It is my belief that this is rarely if ever the case. While it is possible that the deformity is sometimes the consequence of injury received during delivery, the cause of most of the congenital cases goes back to conditions existing before birth. It may be compared to club-foot, and may be due to a faulty position of the child *in utero*, or it may come from more serious conditions, such as malformations, or unequal development of the two sides of the body.

A frequent cause in the acquired cases is irritation of the spinal accessory nerve by an enlarged cervical lymph gland; such is the usual etiology of torticollis following scarlet fever, measles, or diphtheria. I have seen it in the early stage of quinsy, and it may occur in cellulitis of the neck. A cause which the physician should always have in mind is cervical Pott's disease; torticollis may be the earliest, and for several weeks sometimes almost the only, objective symptom of this disease. Torticollis coming on acutely is most frequently due to cold (rheumatism?), occasionally to malaria. I have notes of eight cases clearly traceable to malaria, and have seen at least a dozen others. In the so-called rheumatic torticollis, muscular pain and soreness are rather more prominent than in the other forms.

*Prognosis.*—The result in a case of torticollis depends upon the cause, the severity, and the duration of the deformity. Most of the acute cases recover, under appropriate treatment, in the course of a few weeks, sometimes in a few days. The congenital cases with slight deformity are usually amenable to mechanical or postural treatment if begun early. There is, however, in most of the other varieties a disposition of the deformity, if untreated, to persist, and even to increase. If it has lasted several months the probabilities of spontaneous recovery or even of improvement are small.

*Treatment.*—The first indication is to remove or treat the cause when one can be found. Malarial cases require quinine; rheumatic cases are benefited by rest in bed, hot applications, counter-irritation, friction, and sometimes by anti-rheumatic remedies. Cases which have lasted a month usually require some orthopædic head-support, and those which have lasted six months or more are rarely cured without a surgical operation. This may be either a subcutaneous tenotomy or myotomy of the sterno-mastoid, or an open incision. Whitman gives the result of thirty-two hospital cases, as follows: In seventeen in which the de-

formity had lasted less than six months, ten were cured, the average duration of treatment being three months; four were improved, and three not improved, the average duration of treatment in these cases being eleven months. Of fifteen cases in which the deformity had lasted over six months, none were cured and only six improved, after an average of about eight months' treatment. In the foregoing series of cases the treatment consisted mainly in the use of orthopædic apparatus; later results from incision have been considerably more favourable. But these figures show how serious a matter is an old case of torticollis, and emphasise the importance of resorting to radical measures early in the disease.

#### HYSTERIA.

This is not a disease of childhood, but one which is occasionally seen in early life. All that will be attempted in this chapter is to point out the most common manifestations of hysteria when it occurs in children. After puberty it is essentially the same as in adults.

**Etiology.**—Hysteria is very rare before the seventh or eighth year, and most of the cases seen in children occur after the tenth year. As to sex, there is no such predominance of females as in later life, although even in childhood they are more frequently affected than males. Hereditary influences play an important part in the production of this disease. It is seen in children who inherit a nervous constitution, or in whose parents nervous diseases, such as insanity, or hysteria, or alcoholism have been present. Of the other etiological factors the most important are a disordered nutrition, frequently with anæmia or chlorosis, and overpressure in schools. Masturbation or phimosis may act as an exciting cause, or, indeed, anything which leads to an exalted nervous irritability and depreciation of the general health. It may follow any of the acute infectious diseases; or it may be excited by injury, fright, or imitation.

**Symptoms.**—There is scarcely any disease in which the clinical picture presented is so varied as in hysteria. It may simulate almost any form of organic disease of the brain, lungs, digestive organs, bones, or joints. The most common symptoms may be grouped under four general heads. These are, however, seen in almost every conceivable combination.

1. *Psychical Symptoms.*—When these predominate there may be seen periods of mental depression of longer or shorter duration, a change in disposition, an indifference to surroundings, a capricious humour, or a nervous condition of extreme irritability with irregular paroxysms of laughter or weeping without cause. There may be great excitability of temper, and fits of passion almost maniacal in their severity. There may be various hallucinations. Sleep is frequently disturbed, sometimes by attacks resembling ordinary night-terrors; sometimes somnam-

bulism is present. There is often a disposition to deception about the most trivial matters, which may last for weeks. There is a tendency to imitate the symptoms of various diseases, which the patients may have witnessed in others or about which they have read.

2. *Sensory Symptoms*.—These are the most frequent manifestations of hysteria in early life. There is often general or local hyperæsthesia, which may be so great as to simulate inflammation of the various internal organs. Anæsthesia is much less common, although it may be seen in children as young as eight or nine. Headache is an occasional symptom, and is sometimes associated with great tenderness of the scalp. There may be neuralgias in the different parts of the body, or sharp epigastric pain, sometimes accompanied by vomiting. Sometimes the special senses are affected, giving rise to hysterical blindness or deafness, usually of short duration.

3. *Joint Symptoms*.—These are really a variety of sensory disturbances. They are not uncommon, and are often most puzzling. The symptoms may be referable to the spine, or to any of the large joints, particularly those of the lower extremity. All forms of organic disease of these joints may be simulated. They are usually seen between the ages of ten and fourteen years, and occur in both sexes. There may be lameness referred to one of the large joints, curvature of the spine, or torticollis. The symptoms are most frequently referred to the hip, and next to the knee, the ankle, or the spine. The pain is often acute. It is increased by motion, and by attempts at overcoming the deformity, if any is present. There is a marked hyperæsthesia of the whole limb, and sometimes of the body. In nearly every case there is marked tenderness of the spine upon pressure, especially in the dorsal region. The deformity may be very slight from spasm of the flexors only, or it may be severe, and followed by contracture, so that the thighs may be flexed tightly against the abdomen with the heels against the buttocks. Such deformities may last for months. There may be considerable muscular atrophy, but only that which comes from disuse. A special difficulty in diagnosis arises from the circumstance that these symptoms occasionally follow an injury.

Organic disease of bones and joints may usually be excluded by attention to the following points: The mode of onset is more abrupt than is seen in bone disease, and the course of the disease is quite irregular. The degree of deformity is greater than is seen in bone disease of the same duration. There is general hyperæsthesia of the limb, acute tenderness of the spine upon pressure, and undue sensitiveness to heat or cold. The deformity varies from time to time, being always more marked when examination is attempted. If the patients are closely watched, other evidences of hysteria may be seen. Under complete anæsthesia the contractures may disappear entirely. There is no enlargement



of the articular ends of the bones, no swelling of the soft parts, and no evidence of active inflammation or of suppuration. All the symptoms except the deformity are subjective. Under proper treatment there is in most cases perfect recovery, often in a surprisingly short time.

4. *Motor and Convulsive Symptoms.*—In the milder forms there are seen many varieties of tonic or clonic spasm. There may be local spasm of the eyes, face, or mouth, spasm of the muscles of the neck producing torticollis, of the muscles of respiration causing dyspnœa, which may be constant or paroxysmal. There may be hiccough, or spasm of the larynx causing hysterical aphonia. A very common symptom is hysterical cough, which may be so frequent and so severe—even accompanied by hæmoptysis—that grave disease of the lungs is suspected; the chest, however, is free from the physical signs of disease. There may be frequent attacks of vomiting with eructations; these may be continued sometimes even for months, and in rare instances blood has been vomited. There may be dysphagia from spasm of the œsophagus, or regurgitation of food on attempts at swallowing. In more severe cases we may have the symptoms of chorea major and attacks of hystero-epilepsy. The latter are rare in children and do not differ essentially from such attacks in older patients. There are usually prodromal symptoms. The convulsive movements are exceedingly varied in type. There are painful sensations and sensitive areas, by pressure upon which hysterical symptoms may be increased or even convulsions excited. The respiration may be rapid or irregular. All variations in tonic and clonic spasm may be seen. Opisthotonus is frequent. Consciousness is not fully lost, but is disturbed, and hallucinations are present. The temperature is normal.

Hysterical paralysis is not common in children, but it may be seen even in the very young. Other symptoms occasionally seen in hysteria are persistent anorexia, polyuria, sometimes incontinence of urine, disturbance of the secretion of saliva or perspiration.

The general condition of hysterical patients is usually below the normal. They are poorly nourished and anæmic; they sleep badly; they have capricious appetites and feeble digestion.

**Diagnosis.**—Hysteria is apt to be overlooked because its occurrence in children is not considered as often as it should be. In most cases the diagnosis is easy if hysteria is suspected. A combination of vague disconnected symptoms is usually present which admits of no other explanation. Organic disease can be excluded only by careful and repeated examinations. It is to be borne in mind, however, that hysteria not infrequently complicates organic or constitutional disease. Much importance is to be attached to a family history of hysteria or of other neuroses.

**Prognosis.**—This is better than in adults, especially if the cases are taken in hand early, before the disease has become deeply seated. Very

much depends upon how well the directions for treatment can be carried out. The prognosis is less favourable when the hereditary tendency is strongly marked. In many cases there are relapses later in life.

**Treatment.**—Prophylaxis is of much importance. When a hereditary tendency to nervous diseases exists in a family, or whenever very nervous children are placed under the physician's care, every means should be taken toward muscular development, keeping the nervous system in the background. Such children should lead an out-of-door life as much as possible, preferably in the country. They should keep early hours, have regular exercise, and their education should be directed with moderation and judgment, special attention being paid to regularity of work and the prevention of overpressure in schools. Theatres and exciting books should be avoided. All stimulants, including tea and coffee, should be absolutely forbidden. The diet should be plain and nutritious. It is highly important that such children should be removed from association with an hysterical mother, when this is possible. The best results are usually obtained when the child is taken from his home surroundings and placed in some quiet retreat in charge of an intelligent nurse.

In the general management of a case of hysteria, it is of the first importance that the child should be cared for by a person of firmness, who can exercise proper control. Hysterical children are always managed more easily when they are removed from their homes and placed under the charge of a good nurse. Sometimes they can be managed in no other way. Isolation is absolutely essential in many cases. The general health should be carefully looked after, and arsenic, iron, cod-liver oil, and other tonics given according to indications. Horseback exercise and other out-of-door sports should be encouraged, and every means taken to interest the child in something which requires physical exercise. In cases of simulated disease, the child should be put to bed, no books or toys allowed, and no effort made toward his amusement. No sympathy should be exhibited, but the child should be treated with kindness and firmness. This moral treatment is quite as important as any other part of the therapeutics. In cases with hysterical joint symptoms the most valuable thing is counter-irritation to the spine, preferably by the Paquelin cautery. Under no circumstances should mechanical force be used to overcome deformity. Many cases of hysteria improve under hydrotherapy; the cold douche, the cold pack, or the shower bath may be used. This is valuable in conjunction with massage and the "rest treatment."

#### HEADACHES.

Headaches are not common in little children except in connection with disease of the brain or meninges; in older children they occur from

causes similar to those seen in adult life. The most frequent headaches may be grouped in the following classes:

1. *Toxic Headaches*.—Such are the headaches resulting from uræmia, from malaria, and those seen in many acute infectious diseases. But the largest number are associated with chronic indigestion and constipation.

2. *Headaches from Anæmia, Malnutrition, and Nerve Exhaustion*.—These are most frequently seen in girls from ten to fourteen years old. Some are intellectually bright, and have been crowded in their school work; others are dull and learn only with difficulty, and in consequence worry over their work until their health becomes undermined. They sleep badly, lose appetite, and often become choreic. The anæmia may be either the cause or the result of these symptoms.

3. *Headaches of Nervous Origin*.—These may occur in children who are highly neurotic, either from their inheritance or surroundings, and in those who are the subjects of epilepsy or hysteria, and they may be symptomatic of organic disease of the brain, such as tumour or tuberculous or syphilitic meningitis. True facial neuralgia is rare in childhood except from carious teeth; from this cause, however, it is not infrequent.

4. *Headaches due to Disease of some of the Organs of Special Sense*.—In connection with the eyes there may be conjunctivitis, keratitis, iritis, errors of refraction, or strabismus; connected with the nose there may be polypi, hypertrophic rhinitis, or adenoid vegetations of the pharynx; connected with the ears there may be otitis or foreign bodies in the canal. Each one of these conditions requires special treatment.

5. *Headaches due to Inherited Gout or Rheumatism*.—These are not very frequent, but they may be severe, and may at times simulate the onset of meningitis. They are often accompanied by pains in the joints, muscles, or nerve trunks.

6. *Disturbances of the genital tract* are rarely a cause of headaches in children, although this may be the case in girls about the time of puberty, especially where menstruation is delayed or difficult.

**Diagnosis.**—The diagnosis of headaches includes the discovery of the cause, and this is often difficult. In an infant or a young child, organic disease of the nervous system should always be suspected as a cause of severe headaches. In older children the important things to be considered, because the most frequent, are digestive disturbances, nervous exhaustion, malnutrition, and visual disorders. An absolute diagnosis in a case of persistent headache can be made only by a careful physical examination, not omitting a study of the urine; often there must be a close observation of the patient for some time.

**Treatment.**—The only successful treatment is that which is directed toward a removal of the cause. Each one of the different groups above

mentioned is to be managed differently, according to the principles elsewhere laid down regarding the treatment of these conditions. For the relief of the symptom, cold to the head, a hot foot-bath, and phenacetine in moderate doses are perhaps the most certain of all remedies.

#### DISORDERS OF SPEECH.

In this chapter will be discussed only functional speech defects, those depending upon organic conditions being considered in connection with diseases of the brain. The most common varieties are stuttering, stammering, lisping, alalia, backwardness, and functional aphasia. All forms are much more frequent in boys than in girls, the proportion being more than four to one.

**Stuttering.**—This is the most common form of speech disturbance. Articulation is distinct and the separate sounds are properly produced, but there is a difficulty in connecting the consonant with the succeeding vowel; this seems like an obstacle to be overcome. Occasional stuttering is seen in very many children. It is more frequent in the third and fourth years, before speech is thoroughly mastered. At this age it is aggravated or produced by disturbances of nutrition, but is usually of temporary duration, lasting for a few weeks or months. Only recently a little boy of four was under my care, who became very anæmic, slept poorly, and suffered from malnutrition as a result of the confinement incident to a home in the city. He soon began to stutter, and in a short time it became painfully marked. After a few weeks in the country he improved very much in his general condition, gained four or five pounds in weight, and his stuttering completely, and I think permanently, disappeared. Such disturbances as this are analogous to chorea. In other cases stuttering follows some acute illness, and under such conditions also it is usually of short duration.

Most children who become habitual stutterers do not begin until they are six or seven years old, and sometimes even later. Stuttering may arise from imitation, and inheritance is an important etiological factor. It is frequently a mark of degeneration.

It is important that all such cases receive early treatment before the habit becomes firmly fixed. The prognosis is good for spontaneous recovery in nearly all the cases seen in very young children, and also in those coming on after acute illness. Other cases in which the condition has become habitual should have the benefit of systematic training under a competent teacher in breathing and vocal gymnastics.

**Stammering.**—This term is sometimes used synonymously with stuttering. Kussmaul makes the distinction between them that, in stammering, individual sounds are difficult of production, while in stuttering it is syllabic combinations. Stammering is often accompanied by some

defect in the organs of articulation—the teeth, lips, tongue, or palate—which is not present in stuttering.

The treatment consists in careful training and in the correction of whatever abnormal local conditions may exist.

**Lisping.**—In this there is an imperfect production of certain sounds, owing usually to a faulty position of the organs of articulation. The sounds may be so indistinct that they can not be understood. In this condition also there may be defective formation of some of the organs of articulation, although in the milder forms this is not the case. The treatment is similar to that of stammering.

**Alalia.**—This consists in a total inability to articulate. It is seen in all young infants during their earliest attempts at talking. In older children it is usually associated with some mental defect.

**Backwardness.**—Backwardness is carefully to be distinguished from a late development of speech due to mental defects. At two years old children not deaf are almost invariably able to speak. Speech may be late in consequence of prolonged or very severe illness, and when it has once been acquired it may be lost from similar causes.

**Functional Aphasia.**—The term has been applied to a temporary loss of speech which sometimes occurs in chorea, and sometimes from severe fright or anything else which has produced a marked nervous impression. West records an instance in a girl of eight years, who was suffering from an attack of chorea induced by fright. Speech first became difficult and then was lost altogether. For a month the child could say only “Yes” and “No.” The case improved very slowly, but at the end of nine weeks had recovered completely. Loss of speech sometimes follows the acute infectious diseases, especially typhoid fever.

In all disorders of speech, the functional cases are to be distinguished from those which depend upon deafness and mental deficiency. The frequency with which these disorders are due to disturbances of general nutrition, and to local causes in the mouth and throat, should be borne in mind, and these conditions should receive their appropriate treatment early, before the habit of defective speech becomes firmly established. For the latter class of unfortunates, special training at the hands of a competent teacher should be advised, preferably in an institution.

#### DISORDERS OF SLEEP.

**Disturbed Sleep, Sleeplessness.**—Disturbed or restless sleep is much more common in infancy and childhood than is true insomnia, although the causes of the two conditions may be the same.

**Etiology.**—In infancy these symptoms are most frequently due to hunger or to indigestion resulting from overfeeding or improper feeding. Very often disturbed sleep is the result of bad habits, such as rocking

during sleep or night-feeding. Sometimes it arises from dentition, or the pain of colic or otitis; at other times it may be simply the expression of a condition of extreme nervous irritability, the result of inheritance or of the child's surroundings. It is often caused by the persistent activities of a fussy nurse or mother.

In later childhood the first thing to be suspected when sleep is much disturbed is some derangement of the digestive organs; in this will be found the explanation of fully half the cases. The most frequent type, when the symptom is of long duration, is chronic intestinal indigestion, often associated with indicanuria, a condition in which formerly the usual diagnosis was intestinal worms. Other cases are due to obstructed respiration from adenoid growths of the pharynx or enlarged tonsils, sometimes to nocturnal attacks of asthma. A lack of fresh air in the sleeping room, excessive or insufficient bedclothing, and cold feet, are other frequent causes. Disturbed sleep with "starting pains" is one of the earliest symptoms of hip-joint disease. In the nervous exhaustion resulting from overpressure in schools, and in malnutrition and anæmia, disturbances of sleep are well-nigh constant. They are also seen in organic cardiac disease and in all pulmonary conditions accompanied by dyspnoea or cough. Sleep may be disturbed in consequence of bad dreams which have their origin in exciting stories heard or read just before bedtime, or in too violent or exciting play. To discover the cause in almost any case it is necessary to investigate carefully the whole routine of the child's life.

*Symptoms.*—The condition may be one of real insomnia which may last for weeks or months; or the sleep may be simply disturbed and restless, the child waking many times during the night, and when asleep will not lie quietly, but constantly changes his position. Sometimes children wake suddenly with a scream, but immediately drop off to sleep again.

*Treatment.*—The essential treatment consists in the discovery and removal of the cause of the disturbance. This will often involve a radical change in the manner of feeding, in the hygiene of the nursery, and in all the surroundings of the child. A change of nurses sometimes results in a speedy cure. Under no circumstances should the physician countenance the use of drugs to promote sleep in children, except in the case of severe acute disease. Soothing syrups and all nostrums for "teething" should be absolutely forbidden; also the sucking of a "pacifier." Many mothers and nurses fall into the habit of using them, because the injurious effects are not appreciated. When the cause of sleeplessness is found and removed the child will sleep, but compulsory sleep obtained under other conditions is usually productive of more harm than good. If food, diet, and all bad habits have been corrected, nervous causes should be investigated. When no cause can be discovered the treatment

should consist in putting the child upon the simplest possible diet, and in attention to such general conditions as anæmia, malnutrition, and neurasthenia, some of which are almost certain to be present. In many cases a warm bath at bedtime will be found beneficial. A quiet, darkened room, plenty of fresh air, and the stopping of both eating and drinking during the night, are essential to a cure in most cases. When the condition accompanies some acute disease, the drugs which are most useful are codeine and trional. A child of two years may take gr.  $\frac{1}{10}$  of codeine or two grains of trional as an initial dose, to be increased if necessary.

**Night Terrors—Pavor Nocturnus.**—Two classes of cases have been grouped under this head, both having this in common, that sleep is disturbed by fright.

The condition in the first group partakes of the nature of nightmare. It may be due to partial asphyxia from adenoid growths of the pharynx, or to other causes mentioned under disturbed sleep, or it may be gastric or intestinal in its origin. These cases are quite frequent. Sleep may be disturbed from the outset, and the attack may be merely the culmination of such disturbance. The child wakes in a state of fright and excitement, and often says he has had a bad dream. His mind is clear, he recognises those about him, but it may be a long time before he is sufficiently calm to sleep again. The attack may be remembered perfectly the next day. Cases like this are to be managed in the same general way as those of disturbed sleep above mentioned.

In the second group are the only cases to which the term "night terrors" should really be applied. These are relatively rare, but the condition is a much more serious one. The symptom is generally due to some disturbance of the central nervous system. It occurs especially in those of neurotic antecedents, or those who have previously suffered from infantile convulsions, and it is often the precursor of other nervous attacks—migraine, hysteria, epilepsy, and even insanity. The attack usually comes suddenly where a child has previously been sleeping quietly, and more frequently in the early part of the night than later. He is generally found sitting upright in his bed in a bewilderment of terror, being "afraid of the dog," or "the bear," or there is some other vision or hallucination which has produced the fright. Often this is associated with something of a red colour. The child does not recognise those about him, does not know where he is, and may go to sleep again without coming to full consciousness. The next day there is no recollection of what has happened. Usually no after-effects are seen, but sometimes a large amount of pale urine is passed. The attacks may be repeated at intervals of a few months, or they may occur every few nights; but whatever the peculiar nature of the vision, it is likely to be repeated in nearly the same form. Such attacks have something in common with epileptic seizures, and the diagnosis between them may at times be dif-

ficult. They are to be regarded seriously, not only on account of what they are in themselves, but on account of what may follow.

*Treatment.*—All mental and nervous strain should be most carefully avoided, and when the attacks are frequent the bromides should be given at bedtime. Some person should sleep in the same room with the child, or in an adjoining one with the door open.

**Excessive Sleep.**—It is rare that either infants or children sleep an unnatural amount of the time unless one of two causes is present—organic brain disease, most frequently tuberculous meningitis, or the use of drugs. The latter is always to be suspected if with the sleep there is associated obstinate constipation. Opium in the form of “soothing syrup” or paregoric is the drug which has usually been given.

#### INJURIOUS HABITS OF INFANCY AND CHILDHOOD.

On account of the close connection of such habits with disturbances of the nervous system, they may be properly considered with the functional nervous diseases. Although some of these habits may not be of serious importance, yet as a group they usually receive too little attention at the hands of the physician. The list is very long, and only the most important ones will be discussed.

**Sucking.**—This is a very common habit in infants, and during the first few months it is seen to some degree in most of them. If they are carefully watched the habit is easily stopped; otherwise it may continue indefinitely. Young infants usually suck the fingers when hungry, and this can scarcely be considered abnormal, but an effort should always be made to stop it, lest the habit become fixed. Lindner distinguishes between simple sucking and sucking with combinations. In the former, the child sucks some part of the body, such as the thumb, fingers, toes, tongue, lips, back of the hand or arm, or it may be some foreign substance, such as part of the clothing, the blanket, a rubber nipple, or the “pacifier.” This is the most common form that is seen. In the second variety the sucking is accompanied by the rubbing of some other parts, which seems to afford a pleasurable excitement; this may be the ear, the genitals, or any other portion of the body. Sometimes sucking is accompanied by some practice which produces actual pain, such as pulling of the hair or scratching the body. Habits of sucking often persist throughout infancy, and not infrequently throughout childhood; they have often been known to continue up to puberty. The longer the habit has lasted the more difficult is it to break.

The results of sucking may be serious. Deformities of the thumb or finger, of the lips and teeth, and even of the jaws, are sometimes produced. I knew a woman whose thumbs to advanced age showed a deformity resulting from the habit of thumb-sucking while a child. In



her case the habit was not broken until she was eight or nine years old. Probably the most pernicious result of sucking is its tendency to develop the habit of masturbation. Habitual sucking of one hand or finger may lead to spinal curvature.

*Treatment.*—In the management of these cases the most important thing is to arrest the habit early, before it becomes fixed. Too often the habit of thumb-sucking, or of sucking a rubber nipple, is encouraged by mothers, nurses, and sometimes even by physicians because of the temporary quiet which is thereby produced. Under no circumstance should it be resorted to as a means of putting children to sleep or otherwise quieting the nervous system. With infants, the only treatment which is at all successful is mechanical restraint. It is of no use to cover the part which is sucked with bitter solutions. The hands of young infants may be covered with mittens, or with the long sleeves of a night-gown which is pinned to the bed, so that it is impossible for the child to get the part to the mouth; or, still better, cuffs or splints of pasteboard may be applied at the elbow, so as to prevent flexion of the arms. In the milder cases the habit is often discontinued spontaneously; but when it has been indulged in until a child is four or five years old, it is broken only with the greatest difficulty. Punishments are of little avail, but rewards are often successful.

**Masturbation.**—This is not uncommon even in infancy. Many cases have been observed during the first year, and some as early as the seventh or eighth month. It is seen in children of all ages and in both sexes; but in infants and very young children it is, in my experience, very much more common in girls than in boys.

*Etiology.*—Local causes are present in a large number of the cases, and they are usually something which produces undue irritation. The most frequent are, long or adherent prepuce, phimosis, balanitis, vulvovaginitis, eczema of the labia, threadworms, and tight clothing. A urine which is irritating because of excessive acidity or the presence of crystals of uric acid may be a cause. Any irritation may lead the child to rub the parts in some way, and a pleasurable sensation being excited, this action is repeated until a habit is formed. Other causes are exercises in which the legs are rubbed together, or the body against a pole, as in climbing. To these causes must be added, in infants at least, the habit of sucking. After infancy the habit of masturbation is usually acquired from other children, but sometimes taught by vicious nurses.

General causes are also important as predisposing factors. These are the same as underlie most of the neuroses of childhood—viz., marked anæmia, general malnutrition, and a highly neurotic constitution, which is often an inheritance, and is always aggravated by surroundings which tend to unnatural stimulation of the nervous system. When masturbation develops in a young child without any local cause, it may be an

early sign of either mental deficiency or moral delinquency; if looked for, other stigmata of degeneration will usually be found, and in most cases other vicious traits will soon appear.

*Symptoms.*—In infants and very young children masturbation is usually accomplished by thigh friction or by rubbing the body against a pillow, a chair, or some other object. The variety of ways is almost endless. Frequently the child will simply lie upon the floor with the thighs crossed and rigidly held, and sway the body backward and forward. This lasts for a few moments, is accompanied by flushing of the face and some appearance of excitement, followed by relaxation, and often by perspiration. It frequently happens with little children that these “queer tricks,” as they are often regarded, have been continued for months before their true nature is suspected.

A consciousness that they are doing something wrong early leads even young children to seek seclusion when they repeat the habit. It is especially likely to be practised when children lie long awake alone after they go to bed, or if they wake early. The habit is always made worse by any deterioration of the general health. I have known children, who were thought to be cured, to relapse under such conditions.

It is somewhat difficult to separate the general symptoms with which masturbation is associated, and upon which it largely depends, from those which are the direct result of the habit. There are some children in whom the condition is chiefly or entirely dependent upon a local cause, or when it is only occasionally practised, in whom no general symptoms are seen, or at most only an unnatural shyness and a disposition to seek seclusion. Others are precocious and excitable, with an excessive amount of nervous sensibility. There are others in whom more marked nervous symptoms are present; the most striking are absent-mindedness, loss of power of concentration, loss of interest in all amusements, and mental depression. Some girls of only seven or eight years may have fairly regular periods in which masturbation is practised. In one of my patients such periods for a considerable time occurred monthly. During them even very little girls may lose all sense of modesty or decency. Every particle of self-control is gone. They are passionate, excitable, apparently possessed by the one uncontrollable desire to practise the habit. In the intervals such children may be quiet, modest, sweet-tempered, and perfectly normal. In some older subjects nymphomania, or even insanity, may be the ultimate result. Epilepsy, chorea, or hysteria may develop, particularly where a strong predisposition to them already exists in the family. The effect of masturbation upon the physical and mental development of the child may be serious when it is begun at an early age or is frequently practised. But even more striking is the change sometimes brought about in a child's moral nature. Even little

children of eight or nine years may become centres of moral infection, which may involve a group of playmates or even a whole school.

Local symptoms of masturbation are not always present; in the male there may be redness and slight swelling of the prepuce; the organs may be abnormally large or simply much relaxed. The frequent occurrence of erections in young children is always a suspicious symptom. In the female there is often seen an abnormal development of the genital organs for the age, with an early appearance of pubic hair. No importance is to be attached to adhesions of the clitoris. Sometimes there is vaginitis.

*Prognosis.*—Masturbation in children is at all times a most difficult condition to deal with. The outlook is better in infants and young children than in those who are older, because the latter are more difficult to watch and control; besides, in them the habit has usually become more firmly fixed. In young children local causes are frequently found to be at the root of the trouble; in those who are older general causes are more often present, and these it may be impossible to remove. In almost any case in which the habit has become firmly developed, many months and usually several years are necessary for complete cure. The tendency to relapse is very strong. When masturbation is a symptom of degeneracy it is usually hopeless.

*Treatment.*—The most important thing is an early recognition of the condition. The physician should put parents and nurses on their guard, and the first suspicions should be reported and the child carefully watched until all doubt is removed. In young infants much may be accomplished by mechanical restraint. The kind of restraint which is necessary will depend upon the manner of masturbating. If by the hands, they should be tied during sleep, so that the child can not reach the genitals; if by the thigh-friction, the thighs should be separated by tying one to either side of the crib. In inveterate cases, a double side-splint, such as is used in fracture of the femur, may be applied. In children that are over three years old, all such contrivances are almost invariably unsuccessful. It is of the utmost importance in every case to have the child under the close surveillance of a competent and trustworthy person. He should be especially watched just after being put to bed and immediately after waking. Corporal punishment is often useful in very young children, but of little or no benefit in those who are over three years old. In fact, in such cases it may do positive harm, for deception and lying are soon added to the previous vice. The mother should secure the child's confidence, and in every way possible seek to strengthen his will and stimulate his self-control, using her influence to help him break the habit. The local causes, too, must be examined into and removed whenever found. Circumcision should be done if phimosis exists, and even when it does not, the moral effect of the operation is sometimes of very great benefit. In girls improvement sometimes fol-

lows a separation under anæsthesia of the preputial hood from the clitoris. But unless this is frequently repeated, the adhesions soon recur. Complete circumcision is sometimes done with advantage, and in very obstinate cases the clitoris may be cauterised. Blistering the inside of the thighs, the vulva, or the prepuce is sometimes useful. But as a rule none of these measures accomplishes anything permanent. Care should be taken that the clothing does not irritate the parts. The child should be removed from all vicious companions; but it is quite as important that the greatest vigilance should be exercised in the home and at school, so that the child should have no opportunity to teach other children the habit. In the most serious cases the child should be sent away from home and kept from other children. The co-operation of a trustworthy nurse or companion is indispensable. General treatment should be directed to the child's condition; it is required in most of the cases. A child suffering from malnutrition and anæmia should be sent to the country, kept out of doors and away from books, studies, and from everything which stimulates or excites the nervous system. Almost all exercises except horseback may be recommended. Every means should be employed to build up the general health. These cases are most difficult and most discouraging ones for the physician. A cure results only by using all these measures and for a long time.

**Nail-biting and tongue-sucking** are two forms of habit which are less frequent and less important than those already mentioned. The former is best remedied by wearing gloves and by keeping the nails cut very short. Tongue-sucking seldom becomes a fixed habit, and the child usually ceases it of his own accord as he grows older.

**Pica or perverted appetite** is an inordinate desire to eat various substances, such as dirt, sand, mortar or coal. It is most frequently seen in infants but may occur in older children. This habit is met with in those who are mentally defective, but not rarely in other children. These patients are usually highly neurotic and exhibit some of the other habits common to this class. In some children gastric derangements seem to play the part of an exciting cause. Pica is a common symptom of infection with hook-worm. The habit may continue for years unless corrected. The general health often becomes seriously undermined as a consequence of the disturbed digestion resulting from the presence of abnormal substances in the stomach. Children in whom such a habit is present should in the first place be watched and prevented from indulging in their abnormal craving. Secondly, the digestion and general health should be improved according to indications afforded by the individual case.

**Head-banging** is an expression of extreme nervous irritability most frequently seen in infants or in very young children. It is not indicative of any special form of nervous derangement, but is caused by the same

morbid impulse which leads other nervous children to scratch their faces, pull their hair, etc. While in some children head-banging occurs only occasionally, I have seen patients in whom it existed for a long time. It may be repeated almost every night, and continue at intervals for two or three hours, and that without temper or excitement, but with such force as to produce contusions of the scalp and necessitate padding the sides of the crib. It is rarely a symptom of organic brain disease. Rickets is often associated and the nutrition of most of the patients is much below the normal. The treatment is general.

### CHAPTER III.

#### *DISEASES OF THE BRAIN AND MENINGES.*

##### MALFORMATIONS.

THE malformations of the brain are of great variety, and many of them are solely of anatomical interest, as the conditions are incompatible with life. Only the most frequent and the best-known types will be mentioned, and those which are of interest from a clinical point of view.

**Meningocele, Encephalocele, and Hydrencephalocele.**—These three conditions have in common a protrusion of some part of the cranial con-

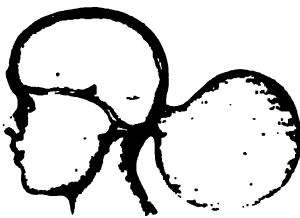


FIG. 106.—MENINGOCELE.



FIG. 107.—ENCEPHALOCELE.



FIG. 108.—HYDRENCEPHALOCELE.

tents through an opening in the skull. In meningocele (Figs. 106, 109) there is protrusion of the membranes alone. These form a sac, which is usually, but not invariably, distended by fluid. In encephalocele (Fig. 107) there is a protrusion of a portion of the brain substance; this is connected with the rest of the brain by a constricted neck or pedicle. The tumour may or may not contain fluid. In hydrencephalocele (Fig. 108) there is a protrusion of a portion of the brain substance which contains within it a cavity filled with fluid, this cavity communicating with the distended lateral ventricles.

In all these conditions there is a tumour, usually pedunculated, of a round or pyriform shape, with a smooth or lobulated surface. The

ordinary size is that of a mandarin orange; it may be as small as a walnut, or as large as the patient's head. It is generally covered by the scalp, which is often denuded of hair; but it may be covered only by granulation-tissue, or it may show a central cicatrix, like that of spina bifida. Other deformities, such as spina bifida, club-foot, and hare-lip are frequently present.



FIG. 109.—MENINGOCELE. From a patient in the Babies' Hospital.



FIG. 110.—FRONTAL MENINGOCELE. From a patient in the Babies' Hospital.

All these conditions are rare, but the most frequent and most serious one is hydrencephalocoele, this being usually associated with hydrocephalus. The next in frequency is encephalocoele, which has the best prognosis. This is frequently termed *hernia cerebri*. If fluid is present, it is external to the brain. In meningocele (Fig. 109) there is simply an accumulation of fluid, which communicates by a small opening with the general arachnoid cavity of the brain.

Of 105 cases collected by Schatz, 59 occupied the occipital region and 46 were frontal. The aperture through which the occipital protrusion takes place is usually in the median line. It may communicate with the posterior fontanel, with the foramen magnum, or with the cleft of a spina bifida. The occipital bone may be divided in the median line, or rarely it may be absent.

In the naso-frontal form (Fig. 111) the tumour is usually at the root of the nose, a little to one side of the median line. The aperture is most frequently between the cribriform plate of the ethmoid and the frontal bones. It may be between the lateral halves of the frontal bone, causing a median tumour. The point of protrusion may also be the lateral region of the skull, generally about the lateral fontanel, or along the line of the sutures; it may project into the mouth or the pharynx. These anterior tumours are usually



FIG. 111.—NASO-FRONTAL MENINGOCELE. Infant one week old.

small, although large ones containing the anterior lobes of the brain have been seen.

The theory of the origin of these malformations which is most widely accepted is that they are primarily cases of intra-uterine hydrocephalus, and as the cranial cavity is gradually closed by the development of the bones, a certain portion of the brain is left outside.

*Symptoms.*—The tumour is always congenital, although after birth it frequently increases very much in size. A typical tumour is round and elastic, usually giving evidence of fluid; it pulsates synchronously with the heart; during screaming or forced inspiration, it increases in size; partial and in some cases complete reduction is possible, but this is usually followed by marked cerebral symptoms, even by convulsions. After partial reduction, an opening in the skull may often be made out. Microcephalus may be present, or there may be unequal development of the two sides of the head.

The following differential points indicate the most characteristic features of the three varieties: In meningocele, the tumour is at first small, but increases; it has a smooth surface; it is pedunculated; there is distinct fluctuation, perfect translucency, rarely pulsation; often it is completely reducible; compression of the tumour causes cerebral symptoms; the skull is normal. In encephalocele, the tumour is small and smooth; it is rarely pedunculated; fluctuation is absent; it is not translucent; there is distinct pulsation; it is usually reducible; pressure causes cerebral symptoms; the skull is normal. In hydrancephalocele, there is a large pendulous tumour with an irregular or lobulated surface; it is pedunculated; translucency is rarely complete; fluctuation is distinct; it is irreducible; pressure rarely causes symptoms; microcephalus and other deformities are often associated.

The occipital tumours are usually more serious than the frontal ones. The majority of cases die in the course of the first few weeks of life, death resulting from meningitis, convulsions, or rupture. In meningocele the tumour usually grows slowly, and ultimately may be shut off from the cranial cavity; but gradual thinning of the membrane may take place, and spontaneous or accidental rupture occur. In encephalocele the tumour grows slightly, or not at all. Most of these patients exhibit signs of mental impairment or other evidences of organic brain disease.

*Treatment.*—According to Treves, operation is justifiable only in case of impending rupture. The conditions present are essentially the same as in spina bifida. Meningocele may be aspirated or the sac may be laid open and a plastic operation performed for the closure of the communication with the cranial cavity; or the skin may be divided, and a ligature or clamp applied to shut off the communication with the brain. All these methods have been at times successful, but recovery has in many instances been followed by the development of chronic

hydrocephalus. Encephalocele is to be treated by protection and compression. Aspiration may be resorted to if fluid is present. In hydrencephalocele the prognosis is absolutely bad under all circumstances. Schatz gives the following statistics, showing the results with and without operation, all varieties being included: Of twenty-four occipital tumours not operated on, three recovered; of thirty-five operated on by excision, ligation, or injection, six recovered. Of forty-six frontal tumours, there were six recoveries in thirty-two cases without operation, and two recoveries in fourteen cases with operation.

**Microcephalus.**—This is often regarded as due to premature ossification of the skull; but the hypothesis is certainly inadequate to explain all or even most of the cases. In many children suffering from marasmus, the sutures ossify and the fontanelles close much earlier than in healthy infants of the same age, chiefly because, with the rest of the body, the brain also has ceased to grow. In microcephalus, I believe it usually to be the case that the early ossification of the skull is due to arrested growth of the brain, and not the reverse. The reasons for the developmental arrest in the brain are for the most part unknown.

It is well known that there is not an invariable relation between the size of the head and the size of the brain, although generally the two correspond. If the circumference of the head is much below the average for the age (see introductory chapters), and relatively much less than the measurements of the rest of the body, microcephalus may be assumed to exist. Sachs calls attention to the fact that the circumference of the head may be nearly normal and yet the essential conditions of microcephalus exist, owing to imperfect development of the anterior part of the brain.

The symptoms of microcephalus are those of mental deficiency and cerebral paralysis, existing in all possible combinations and with variable degrees of severity.

The essential condition in microcephalus being an arrest in the development of the brain, it is not difficult to understand why the operation of craniectomy once thought so promising has been generally abandoned. The results do not justify any other operative measures yet proposed for the relief of these cases.

**Congenital Hydrocephalus.**—These cases may fairly be considered as belonging in this group, although they are discussed elsewhere.

**Porencephalus** (literally, a hole in the brain) is a condition in which there is a large depression in some part of the brain, but with surrounding parts well developed. Such depressions may involve a whole lobe, and they may be deep enough to reach the lateral ventricles.

Porencephalus is described as congenital or acquired. In the congenital form, the defect is usually found in the anterior or middle part of the brain. The origin of these conditions is still a disputed question.



They are probably due to early vascular changes. Children sometimes live several years with very large defects, the symptoms depending upon the seat of the lesion. The acquired form of porencephalus is usually one of the late results of meningeal hæmorrhage. It may affect one or both sides. Such cases present the symptoms of spastic paralysis—usually diplegia. In all cases with large brain defects, the space is filled with fluid.

#### PACHYMENINGITIS.

Pachymeningitis, or inflammation of the dura mater, occurs both as an acute and a chronic disease.

**Acute Pachymeningitis.**—This is very rare in children. Only pachymeningitis externa is generally included under this term, as acute pachymeningitis interna does not occur alone, but usually with inflammation of the pia mater (leptomeningitis). It may be associated with disease or injury of the bones of the skull, but is most frequently seen in connection with middle-ear disease. It generally begins as a localised process, but the inflammation may extend to the inner layer of the dura, and to the pia mater; or it may remain circumscribed, and terminate in the formation of an abscess between the dura mater and the bone.

The symptoms of acute pachymeningitis are distinctive only when the process is localised. They are then usually associated with middle-ear disease, and are indistinguishable from those of cerebral abscess. The treatment is surgical.

**Chronic Pachymeningitis.**—This, in children, almost invariably affects the inner layer of the dura mater (pachymeningitis interna); it is also known as *pseudo-membranous* and as *hæmorrhagic pachymeningitis* or *hæmatoma of the dura mater*. Its causes are for the most part unknown. It is a rather rare condition, being usually discovered at autopsy in children, chiefly cachectic infants, who have died of other diseases.

Two classes of cases are to be distinguished—those with, and those without extensive hæmorrhages. In the latter group there is found a thin, translucent, vascular membrane lining the inner surface of the dura. It may be only a delicate film which can be scraped off; it may be as thick as ordinary blotting-paper, or even twice that thickness. The membrane is often œdematous; it is exceedingly vascular, and the vessels have very thin walls. There are usually scattered punctate hæmorrhages, and there may be a few of larger size. This membrane may cover the whole inner surface of the dura, but in most cases it is principally over the convexity and may be found only here; it is apt to be more upon one side than upon the other. In cases of long standing there may be adhesions between the dura and the pia. When large hæmorrhages have taken place, quite a different pathological appearance is presented. The lesions found in a case upon which I made an autopsy in the New

York Infant Asylum are fairly typical: The infant was six months old, and the symptoms had existed for six days. The fontanel was bulging to a marked degree, and the sagittal and coronal sutures were separated. A thin recent clot from one-eighth to one-fourth of an inch in thickness covered nearly the whole of the right hemisphere and part of the convexity of the left. The entire dura was lined both at its convexity and base by a pseudo-membrane of grayish color, about one-sixteenth of an inch in thickness. The brain was anæmic.

In cases of longer standing partial organisation of the clot may be seen; in more recent ones the blood is partly or entirely fluid. I once found acute leptomeningitis with a purulent exudation, associated with hæmorrhagic pachymeningitis. In cases where life is prolonged for years, there may be partial or even complete absorption of the clot, followed by the formation of cysts, considerable inflammatory thickening of the pia with deposits of blood pigment, and finally atrophy and sclerosis of the cortex. The source of the hæmorrhage may be the rupture of a single large vessel, but more frequently the blood comes from many small vessels.

*Symptoms.*—These are due to the hæmorrhage, and not to the inflammatory process. Until hæmorrhage occurs there are no symptoms by which the disease can be recognised. Thus in many of the cases in which pachymeningitis is found at autopsy, its existence is not suspected during life. The occurrence of hæmorrhage is sometimes marked by vomiting or convulsions, and usually there is loss of consciousness. It may be a question whether the convulsions are the cause or the result of the hæmorrhage. In most cases they seem to be the result. They are usually general and repeated. If the hæmorrhage occurs slowly, there may be stupor without convulsions until nearly the close of the disease. In the fatal cases the symptoms generally continue from two days to a week. There are dulness, stupor, and finally coma, death occurring in coma or convulsions. If the hæmorrhage is diffuse—and this is apt to be the case—there is rigidity of all the extremities; if it is of one side only, the rigidity affects only one arm and leg. The pupils are more frequently contracted, but may be dilated or unequal. There is diplegia, hemiplegia, or monoplegia, according to the seat and extent of the hæmorrhage. The respiration is slow and irregular and may be of the Cheyne-Stokes variety. The pulse is slow, irregular, and sometimes intermittent. The temperature is at first normal, but rises slowly until death occurs, when it is from 100° to 103° F. Generally the cranial nerves are not affected, and opisthotonus is absent. The knee-jerk is often exaggerated. In cases which do not prove fatal—these being chiefly in older children—we have a similar onset, but after a few days consciousness is regained, and only hemiplegia or monoplegia remains. The course of the paralysis is that seen after meningeal hæmorrhage.

due to other causes. Wagner has reported a case in which recurring hæmorrhages took place at intervals of several months, the autopsy showing distinct evidences of both old and recent lesions.

Pachymeningitis, I am inclined to believe, plays a much more important rôle in the production of meningeal hæmorrhages in children than has generally been accorded to it. From the frequency with which this lesion is found as a cause of sudden meningeal hæmorrhages which are fatal, it is not unlikely that some of the cases which recover with hemiplegia or monoplegia, may be due to the same cause.

The prognosis depends upon the age of the patient and the extent of the hæmorrhage. Extensive hæmorrhages are usually fatal in infancy, but small ones are seldom so, for they are rarely at the base. The prognosis of the paralysis in cases not terminating fatally is the same as after meningeal hæmorrhage due to other causes, with perhaps an added liability to recurrent attacks.

Without large hæmorrhages, pachymeningitis interna can not be diagnosed; and it is impossible to differentiate the hæmorrhagic cases from other varieties of meningeal hæmorrhage. It is important to make a diagnosis between pachymeningitis with hæmorrhage, and acute simple meningitis. In the former there is a sudden onset; stupor occurring early, usually on the first day, gradually diminishing in cases of recovery, or deepening into coma in fatal cases; localised or general paralysis, also occurring early; there is no fever in the beginning, and only moderate fever at the close. In acute meningitis there is usually a higher temperature, especially early in the disease; coma develops later, and rigidity of the extremities is less pronounced. In certain cases, however, when the hæmorrhage occurs in the course of some other disease, a differential diagnosis may be impossible.

*Treatment.*—The treatment of pachymeningitis hæmorrhagica is symptomatic. The indications are, to relieve cerebral congestion by applying ice to the head, to allay irritative symptoms by the use of bromides, and to keep the patient perfectly quiet.

#### ACUTE MENINGITIS.

Several different varieties of acute meningitis are met with in children. Cerebro-spinal meningitis is the only form which occurs epidemically; but this is also seen as a sporadic disease. It is due to a specific organism, the meningococcus. There are several other forms of acute meningitis which more or less closely resemble cerebro-spinal meningitis clinically, and which were for a long time confounded with it. Pneumococcus and influenza meningitis are usually secondary inflammations, but sometimes are apparently primary. The typhoid bacillus and the gonococcus may cause acute meningitis, but very rarely in children.

Acute meningitis may be due to any of the pyogenic organisms. This is sometimes spoken of as "septic" meningitis, and is almost invariably secondary. Finally, there is tuberculous meningitis, altogether the most common variety in young children except during epidemics of cerebro-spinal meningitis.

Some idea of the relative frequency of the different forms of acute meningitis as seen apart from epidemics, may be gained from the following figures which give the number of cases occurring in the Babies' Hospital for a series of years, the diagnosis in every case being made by lumbar puncture or by autopsy. The patients were nearly all under three years of age. The organism found was as follows:

|                                      |            |
|--------------------------------------|------------|
| Tubercle bacillus.....               | 157 cases. |
| Pneumococcus.....                    | 23 "       |
| Meningococcus (sporadic).....        | 24 "       |
| Staphylococcus or streptococcus..... | 11 "       |
| Influenza bacillus.....              | 5 "        |
| Colon bacillus.....                  | 1 "        |

#### CEREBRO-SPINAL MENINGITIS.

(*Epidemic Meningitis; Cerebro-spinal Fever.*)

Epidemics of cerebro-spinal meningitis are separated by quite long intervals and occur without any assignable cause. The following chart (Fig. 112) represents the prevalence of the disease in New York City during forty years. But little was seen of cerebro-spinal meningitis until the epidemic of 1872. Since that time a certain number of deaths from

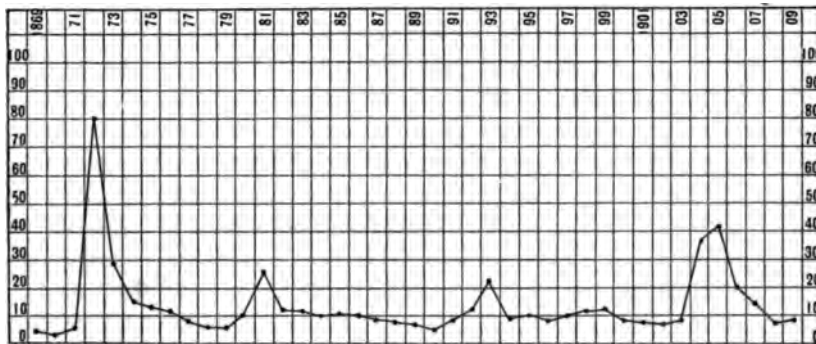


FIG. 112.—CHART SHOWING DEATHS FROM CEREBRO-SPINAL MENINGITIS IN NEW YORK CITY, FOR FORTY YEARS, PER 100,000 OF POPULATION.

this cause have occurred each year; but there have been seen about once in ten years epidemics of greater or less severity. The most important one was that of 1904-5. After each epidemic, for two or three years, the disease is prevalent, but it occurs with gradually lessening frequency until the average incidence is reached. What has been said of New

York is true of almost every large city. In remote country towns, epidemics are occasionally witnessed, and after prevailing a few months the disease disappears as mysteriously as it came. Epidemics are usually seen in the winter and early spring, lasting for several months, generally reaching their height in March or April and slowly subsiding as warm weather approaches.

With reference to the cause of epidemics very little is known. When the disease prevails in cities it occurs especially in crowded tenements, being relatively infrequent in private houses.

Cerebro-spinal meningitis has only recently been included among the communicable diseases. In a series of observations made by the New York Health Department the meningococcus was found in the nasal secretion of fifty per cent of the cases of meningitis examined during the first two weeks of the disease. It was found in the nasal mucus in ten per cent of the persons in close contact with cases. In Flexner's experiments upon monkeys he found the organism in the nasal mucus after animals had been inoculated by way of the spinal canal. These observations indicate that the nasal mucosa is a common avenue of infection and probably also a channel of elimination. The degree of communicability when compared with the common contagious diseases seems very slight. In fully seventy per cent of the cases investigated in the New York epidemic of 1904-5, but one person in a household was affected, although no effort at isolation was made. I have never known the disease to originate in a hospital patient, although in New York cases of cerebro-spinal meningitis have been until very recently received into the general wards with other patients. Sporadic cases of meningitis occur after epidemics, and quite apart from them without apparent cause, and it is very exceptional that any connection with a previous case can be established. About fifty per cent of the cases of cerebro-spinal meningitis occur in children under five years, and about twelve per cent in those under one year. The youngest case I have seen was in an infant six weeks old.

The specific organism of cerebro-spinal meningitis is the diplococcus intracellularis of Weichselbaum or, as it is now generally designated, the meningococcus. It is present in the meningeal exudate, in the cerebro-spinal fluid obtained by lumbar puncture, and in some cases can be demonstrated in the blood, the lungs, and other organs, sometimes in the large joints. It is almost invariably found in pairs or tetrads within the leucocytes. It is decolourised when stained by Gram's method. Outside the body the organism is unknown.

**Lesions.**—In epidemic meningitis death may take place so early that the changes found at autopsy are slight. There may be only a serous exudation and intense hyperæmia, which is doubtless much less marked after death than during life. The cerebro-spinal fluid is turbid and

much increased in amount. The microscope, however, may show, even in these early cases, an abundant exudation of leucocytes in the pia mater. After the third day the lesions are quite uniform. The convolutions appear somewhat flattened from pressure due to distention of the ventricles. The inner surface of the dura is usually normal or only congested. There may be thrombi in any of the cerebral sinuses, or in the meningeal veins of the convexity. There is an exudation of greenish-yellow fibrin, which is sometimes very abundant. It is generally widely distributed, but is most marked over the anterior half of the brain and at the base. In some cases it is limited to the base, but very rarely limited to the convexity. There is an increase in the quantity of cerebro-spinal fluid. The ventricles are moderately distended with serum or sero-pus, and their walls may be slightly softened. The brain substance of the cortex may be reddened or may appear normal. In the meninges of the cord, lesions similar to those of the brain are usually seen. The exudation is principally upon the posterior surface, and may extend throughout the entire length of the cord, or be limited to its upper or to its lower portion.

Microscopical examination shows the exudation to consist of fibrin and pus cells, which infiltrate the pia mater. The superficial layers of the cortex in the inflamed areas often show minute hæmorrhages and very marked cell-infiltration. Minute abscesses may be present. Very marked degenerative changes can usually be demonstrated in the nerve cells themselves. The cells of the neuroglia are also affected; they are swollen and increased in number; and there may be proliferation of the connective tissue about the blood vessels. Changes similar to those just described may be found in the cord, but these are less frequent and as a rule much less severe than those in the brain. Inflammatory products are sometimes present in the central canal of the cord and in the walls of the lateral ventricles of the brain. The inflammatory process frequently extends along the cranial nerves, especially the auditory and optic, and this may result in otitis or choroiditis: from the cord, it may extend along either the anterior or posterior nerve roots. Descending degeneration is found in the nerves both of the brain and cord.

In patients that die after the disease has lasted two or three months, the later results of these lesions may be seen. There is usually present a chronic meningo-encephalitis, sometimes diffuse, sometimes localised. The pia mater is cloudy, thickened, and frequently adherent to the brain. Here and there are seen small, yellow, opaque patches which are the result of fatty changes in the cells and fibrin of the exudate, with some proliferation of connective tissue. The lesions are usually most marked at the base, where the thickening of the meninges and the adhesions may lead to the development of a secondary hydrocephalus.

In cases which have lasted a much longer time the most marked

changes are in the brain substance. There may be generalised meningeal adhesions,<sup>1</sup> with a diffuse cortical atrophy, but more frequently there are areas of sclerosis, especially over the frontal and temporo-sphenoidal lobes, with which there are almost always associated marked descending degenerative changes in the cord. Such lesions are, of course, permanent, and seriously interfere not only with the functions, but also with the growth and development of the brain.

The visceral lesions most frequently found in epidemic meningitis are pulmonary. There may be lobar or broncho-pneumonia, and in the lungs may be found the same organism as in the brain. Acute degeneration of the liver and kidneys is also frequent. The other viscera are seldom affected. Occasionally suppurative inflammation of the joints occurs.

**Symptoms.**—The symptoms of cerebro-spinal meningitis do not differ essentially in the sporadic and epidemic cases, except that the most severe forms of the disease are seen in the latter. They may be divided into several quite distinct groups:

1. *Hyper-acute Form.*—Cases of this kind are rarely seen except in an epidemic, and usually occur at its height. The onset is very abrupt, the course short and intense, and death may take place in from twelve to thirty-six hours. The following case illustrates this type: A little girl of ten years was well enough at 2 P.M. to carry a bundle of clothes a dozen city blocks. Returning home, she complained of intense headache, vomited frequently, and was so weak that she was obliged to go to bed. In a few hours she passed into deep coma, with very high fever, and died at 11 P.M.

The earliest symptoms are usually intense headache, repeated attacks of vomiting, and very high fever. There is great prostration and the nervous symptoms increase so rapidly that in a few hours the patient may become comatose and death occur in a short period. The temperature rises rapidly to 103° or 104°, sometimes to 106° F. A few petechial spots may be discovered over the face, chest, or extremities. There is usually no rigidity, but rather general relaxation. The pulse is weak, in most cases rapid, but sometimes slow and irregular. The respiration is usually irregular both in frequency and depth.

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<sup>1</sup> This lesion and its effects are well illustrated by one of my own patients who died six months after an attack. She was a bright little girl of four and a half years, and had a typical attack of meningitis of moderate severity. Convalescence was slow, but at the end of two months recovery was perfect in everything but her mental condition. She remembered nothing which she had previously learned in the kindergarten, where she had been an exceptionally bright pupil. Her mind was a blank. She was dull, listless, and her face had a vacant, idiotic expression. The special senses seemed unaffected, and her speech was retained. She died during an attack of convulsions. At the autopsy the pia was everywhere thickened and adherent, while in the cortex were present the earlier changes of a general encephalitis.

The symptoms appear to be due to two factors: First, the intensity of the infection; second, the rapid accumulation of cerebro-spinal fluid, causing coma with cardiac and respiratory paralysis. Usually both these factors are present, but I believe that the second one is the more important. In support of this view is the striking infrequency of cases of this type in infants with an open fontanel. Should the patient survive the violence of the onset, a period of reaction occurs, and after a day or two the disease follows the regular course.

2. *Usual Form.*—In this also the onset is generally abrupt, but not so violent as in the cases just described. It may be marked by intense headache, vomiting, convulsions, delirium, chills, and fever with general hyperæsthesia and rigidity. The initial temperature is from 101° to 104° F. Opisthotonus, with severe pains in the back of the neck and along the spine, and general muscular rigidity are usually present. There is often active delirium, but rarely stupor or coma. The pulse is generally rapid, 120 to 150, and sometimes irregular. The respiration is often slightly irregular, and it may be rapid or slow. The eruption is not so frequently seen as in the very acute cases.

As the disease progresses, the nervous symptoms often change but little from day to day for two or three weeks. They are mainly of the



FIG. 113.—POSTURE IN CEREBRO-SPINAL MENINGITIS. (Smith.)

irritative type—moderate delirium, extreme hyperæsthesia, tremor and muscular rigidity. The posture is quite characteristic (Fig. 113). Owing to the opisthotonus the child can not lie upon the back, but rests upon the side, with arched spine and neck, and general flexion of the extremities. There is a rather rapid loss in weight, steadily increasing prostration, and a weak, rapid pulse. The bowels are usually constipated. From time to time attacks of vomiting occur. In most cases there is considerable difficulty in feeding. The duration of this form of the disease is from three to six weeks. The course is often marked by periods of remission and exacerbation. If recovery is to take place, the tem-



perature gradually falls to normal and often at times it is subnormal. The mind becomes clear, and one by one the nervous symptoms disappear, the muscular rigidity being usually the last to go. Convalescence is always protracted.

In cases ending fatally, the patient usually passes into a deep stupor or coma, with extreme prostration, a slow, weak, irregular pulse, shallow respiration of the Cheyne-Stokes variety, sunken abdomen, general relaxation, and death occurs from exhaustion or from broncho-pneumonia.

Occasionally the attack is much prolonged, the fever and all the active symptoms continuing from eight to twelve weeks. Emaciation sometimes becomes extreme, and with a few nervous symptoms may continue long after the fever ceases. In infants, death is often due to marasmus. While a fatal outcome is more frequent in these prolonged cases, not a few recover completely, even when symptoms have lasted for eight or ten weeks.

3. *Mild Form*.—Especially toward the end of an epidemic, and sometimes occurring sporadically, there are seen cases which in their onset and for the first two or three days resemble those just described; but instead of running the usual course, the fever and the nervous symptoms subside rapidly and convalescence is established early.

4. *Chronic Form*.—Owing sometimes to the extent, sometimes to the position of the lesions, the disease does not subside at the usual time, but nervous symptoms continue after the temperature and most of the other constitutional symptoms have passed away. These cases are chiefly of the basilar type, and often lead to the development of chronic basilar meningitis with secondary hydrocephalus. They are more fully considered in a later chapter.

*Onset*.—One of the most striking features of this disease is the abruptness with which it develops. Occasionally there are indefinite symptoms for a day or two before active symptoms begin; but in the great majority not only the day, but the hour of the onset is definitely marked. The most frequent initial symptoms are the simultaneous occurrence of severe headache and vomiting, followed by high fever and marked prostration. The vomiting is usually repeated, projectile, and has no relation to meals. Convulsions occurred in the beginning of thirty per cent of my cases. Occasionally a decided chill is seen. After twenty-four hours acute general pains and hyperæsthesia are usually present, together with rigidity of the muscles of the neck and extremities, giving rise to opisthotonus and muscular contractions.

*Skin*.—Eruptions upon the skin vary much in frequency in different cases and in different epidemics. The most characteristic one is the appearance of small punctate hæmorrhages, resembling flea bites; they are not numerous, but may be found on almost any part of the body, most frequently upon the extremities, the upper part of the chest, and

neck. In my experience they have been present in about fourteen per cent of the cases. Sometimes larger hæmorrhages are present. From this symptom the name "spotted fever" has arisen. This petechial eruption belongs to the early stage of the disease, fades quickly, and is rarely visible after the third or fourth day. In some cases a general erythema is present; in others, an eruption closely resembling measles. Herpes upon the lips and face is common in older children, but is rare in infants. Bed-sores have been seen in about one-third of my cases. They are found over pressure points—the trochanter, the malleoli, and the side of the head; in several instances the ear has been the part affected.

*Nervous System.*—Headache is a frequent initial symptom and is usually severe; it is more often frontal than elsewhere, and may be associated with vertigo. There are acute pains in the back of the neck, along the spine, and marked general hyperæsthesia, which is often so intense that any movement of the body causes agonising cries. This is one of the most striking symptoms of the disease, and may continue throughout the acute stage. The mental state varies much in different cases. Delirium is frequent in the early stage of the severe form; it is usually wild and active. After delirium a stage of dulness or apathy ensues, giving place to great irritability when the patient is disturbed. Convulsions are sometimes seen early, but are seldom repeated in the course of the disease or toward its close. There is rarely continuous or deep coma except toward the end of fatal cases. In some cases with high temperature and quite severe symptoms, after the subsidence of a short early stage of excitement or delirium, the mind remains perfectly clear throughout the attack. Under these circumstances an erroneous diagnosis is often made, particularly if the physician has not observed the case from the beginning.

Tonic spasm of the various muscular groups is one of the most characteristic features of this disease and is seldom absent. Like the hyperæsthesia it is persistent. The rigidity and contraction of the muscles of the neck and back produce cervical or general opisthotonus; cervical opisthotonus is most marked with lesions chiefly at the base, and may be wanting in the rare cases when the lesion is almost entirely at the convexity. Tonic spasm of the extremities usually causes general flexion of the thighs, legs, and arms. Late in the disease this may be replaced by complete extension of the lower extremities with dropping of the feet. The tonic muscular spasm gives rise to Kernig's sign, viz., inability to extend the leg when the thigh is flexed upon the body. In young children one should not place too much dependence upon this sign. While rarely wanting in cerebro-spinal meningitis, it is often present in other conditions. Muscular rigidity is one of the most common symptoms and one of the last to disappear. It may be absent in the early

stage of the hyper-acute cases, and very late in fatal cases, when there may be general relaxation. Other nervous symptoms frequently present are ankle clonus, muscular tremor, especially of the hands, and paralysis, which may be facial, monoplegic, or hemiplegic. Early in the disease the knee-jerks are usually increased; in the later stages they are often lost.

*Eye and Ear.*—The pupils in the early stage are generally contracted; toward the close they are usually widely dilated. Ocular paralyses are not so frequent nor so marked as in tuberculous meningitis. The same is true of the changes in the optic disc, although these vary much in different epidemics. There may be congestion of the fundus, retinitis, or optic neuritis. In some epidemics such changes have been observed in fully half the cases. In that of 1904–5, in my own hospital cases, they were rarely seen, and then were but slightly marked. Conjunctivitis is frequently present and may be severe. There may be choroiditis and sometimes complete destruction of the eye, but usually this is unilateral. In most epidemics the ears are more frequently affected than the eyes. Early deafness may be due to a lesion of the auditory nerve, is generally bilateral, and often permanent. Acute otitis media occurs as a complication, and the meningococcus is occasionally found in the discharge. This was true of three of my hospital cases. Permanent deafness is sometimes due to changes in the brain itself.

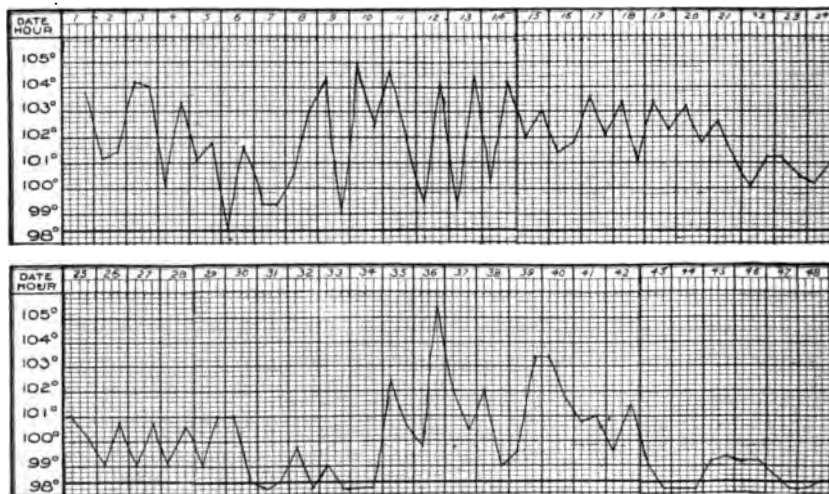


FIG. 114.—CEREBRO-SPINAL MENINGITIS. Recovery. Fairly typical chart of prolonged case, showing remissions and exacerbations. Private patient, three and a half years old; unconscious, blind, and deaf for two and a half months; complete recovery.

*Fever.*—This disease is usually attended by high fever, but the curve is apt to be an irregular one and show wide variations. The temperature is nearly always high at the onset; in the hyper-acute cases it may

reach 106° F. or higher. The usual range during the disease is from 100° to 105° F. (Fig. 114). Sometimes it is steadily high; not infrequently a few days after a sharp acute onset it falls nearly or quite to normal and remains there for several days. Cases seen in this afebrile period are most difficult of diagnosis. This stage may be followed by another sharp rise, and afterward continuous fever. Periods of remission and exacerbation in the temperature are seen in a large proportion of the prolonged cases. Often it becomes subnormal. The temperature may bear no relation to the severity of the other symptoms. Its course is greatly modified by the serum treatment.

*Respiration* is disturbed very early in the disease, when it is often irregular and may be slow or rapid. Throughout the greater part of the attack it may be nearly normal. Occasionally it is of the typical Cheyne-Stokes variety.

*Pulse*.—Throughout the greater part of the disease the pulse is rapid. In the early stage it is often weak, and sometimes irregular. The average frequency in young children is from 130 to 150. A slow, irregular pulse is occasionally seen late in the disease in patients who are in deep coma.

*Blood*.—A leucocytosis is present in nearly all cases. The average is from 15,000 to 30,000. The increase is chiefly in the polymorphonuclear cells. Blood cultures made early in the disease have in some cases shown the presence of the characteristic organism.

*Digestive System*.—Vomiting is one of the most frequent symptoms of onset but rarely persists throughout the attack. Late in the disease it may be most troublesome. As a rule constipation is present. The tongue is coated, dry, glazed, sometimes covered with sordes. In a small proportion of cases jaundice has been observed. On account of the loss of appetite, great irritability, delirium, and stupor, the greatest difficulty is often experienced in feeding these patients. In young children gavage is much more satisfactory than rectal feeding. Early in the disease the abdomen is natural. In the late stage it is often very much retracted.

*General Nutrition*.—This is impaired in nearly all cases. There is a progressive wasting, greater than would be explained by the disturbance of digestion. In the protracted cases it may be extreme. Infants and young children often die of inanition or marasmus long after the active symptoms of the disease have subsided.

Other symptoms of importance are the tense, bulging fontanel, in infants rarely absent early in the attack, but often wanting in the late wasting stage; incontinence of urine and fæces, and retention of urine, very frequent and often overlooked; occasionally swelling of some one of the large joints is seen.

*Course, Duration, and Termination*.—Excluding the fulminating cases in which death occurs very early, the usual duration of active symptoms

in cases not treated with serum is from three to six weeks. Of 350 cases recovering without serum, the disease lasted less than one week in three per cent; in fifty per cent it was five weeks or longer. Some very protracted cases terminate favourably. I have seen one child recover completely after 84 days of fever, and another after 102 days. Most of the prolonged cases are marked by periods of exacerbation and remission. Not until the temperature has been normal for several days, the mind has become clear, and the hyperæsthesia and rigidity have entirely disappeared, can we consider convalescence as established. Recovery is slow, and it may be many months before the child is quite well. In 220 cases receiving serum treatment the average duration of active symptoms after the first injection was 11 days.

In fatal cases, death may come early from coma, convulsions, or heart failure. It may occur in the middle period from complications, most frequently pneumonia, or the terminal stage of the disease may be seen with extreme wasting, and finally death from exhaustion.

**Complications and Sequelæ.**—The chief ones are pneumonia, otitis, conjunctivitis or choroiditis, and bed-sores. Rarely, nephritis and arthritis are seen. Sequelæ are, unfortunately, very common. There may be perfect recovery so far as physical functions are concerned, but the child be left mentally deficient. In some cases the defect is so slight as not to be evident for several months or even years; in others the mental faculties are entirely lost. There may also be various types of paralysis—strabismus, facial paralysis, monoplegia, hemiplegia or diplegia, and often contractures, which are sometimes temporary, but apt to be permanent. The acute attack may be followed by chronic meningitis with hydrocephalus. Deafness is quite common, usually of both ears, and deaf-mutism is not an infrequent result in young children. Blindness is not so common and is usually unilateral. As a late result epilepsy may develop.

**Prognosis.**—The mortality is usually higher in epidemics than when the disease occurs sporadically. It is usually greater at the height of an epidemic and lower at its close. The average mortality before the serum treatment was about 70 per cent. I know of no epidemic on record in which the mortality was less than 50 per cent. In the last year (1905) of the New York epidemic, of 1,780 cases tabulated by the Department of Health the mortality was 76 per cent. Of 59 cases treated in my hospital wards in the same epidemic the mortality was 80 per cent, nearly all these patients being under three years of age. Of 24 cases under one year only one recovered. Of the cases I saw in private practice, largely older children, the mortality was 50 per cent. Not all of those who do not die are to be classed as recoveries, for in fully 25 per cent serious sequelæ remain. The results with Flexner's serum are referred to under Treatment.

**Diagnosis.**—Lumbar puncture is the only accurate means of diagnosis we possess. By it we can not only differentiate meningitis from other diseases with nervous symptoms, but can distinguish this from other varieties of meningitis. Furthermore, this is possible very early in the disease. With proper precautions I believe it to be practically free from danger, and it should be employed whenever meningitis is suspected. The procedure is not difficult, but the technique is important.<sup>1</sup> The quantity of fluid which may be removed at one time varies from a few drops to three or four ounces. During the first day or two it is usually a slightly cloudy or turbid serum; sometimes it is thick and purulent. As the disease progresses the pus cells gradually diminish, and in favourable cases disappear, but may reappear with an exacerbation of the symptoms. These changes are much modified by serum injections.

The presence of many leucocytes in the cerebro-spinal fluid indicates meningitis, which may be due to the meningococcus, but also to the pneumococcus, the influenza bacillus, the staphylococcus, or the streptococcus. The variety can be determined only by microscopical examination of stained smears from the sediment of the fluid obtained after standing or after centrifuging, and by cultures, which should be made immediately after the fluid is withdrawn. In cerebro-spinal meningitis diplococci are found within the pus cells and some are also free in the fluid. The organisms are usually numerous.

The diagnostic value of lumbar puncture, when properly performed, is very great; not only are positive findings conclusive, but early negative findings almost certainly exclude meningitis. I have met with two exceptional cases in which early punctures gave a clear fluid and no organ-

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<sup>1</sup> Puncture may be made with an ordinary surgical exploring needle, but the special lumbar needle devised by Quincke is preferable. This is merely a fine trocar and cannula and is made somewhat stronger than an exploring needle, which sometimes breaks. The child is placed upon the right side with the thighs tightly flexed against the abdomen to separate the spines and laminae of the vertebrae as much as possible. The point chosen for puncture is in the median line between the third and fourth lumbar vertebrae. This is on a level with the highest part of the iliac crest. The strictest asepsis is required. The skin should be carefully cleansed and the needle boiled. The pain is no greater than from exploratory punctures elsewhere. No anæsthetic is necessary for infants, but sometimes is required for older and especially sensitive or nervous children unless they are comatose. Local anæsthesia may be employed or a few whiffs of chloroform given, but always with caution, for the combined shock of the puncture and the chloroform is sometimes considerable. The child should be closely watched for at least fifteen minutes after the puncture is made. The canal is reached at the depth of about one inch. The trocar is now withdrawn and the fluid usually flows freely through the cannula, sometimes spurting forth some distance, owing to high pressure. A dry puncture is generally due to the fact that the canal has not been entered; sometimes that the exudate is too thick to flow through the small needle, or that the needle has been plugged. Raising the patient to a sitting posture usually causes a freer flow, as does also flexing the head upon the chest if opisthotonus is extreme.

isms were found; a few days later the fluid was turbid and organisms were abundant. The meningococcus may persist for a long time. In one of my cases not treated by serum it was present on the ninetieth day.

The diagnosis of cerebro-spinal meningitis by symptoms alone presents peculiar difficulties at the beginning of the attack. The most valuable early symptoms for diagnosis are, a sudden onset with intense headache, vomiting, high temperature, prostration, the petechial eruption, marked rigidity of the neck and extremities, with hyperæsthesia, great irritability or early stupor, even coma. Later, three symptoms are rarely wanting—persistent hyperæsthesia, muscular rigidity of the neck and extremities, and fever. Kernig's sign is seen in other conditions and is not diagnostic. The spinal symptoms are more to be relied upon for diagnosis than are the cerebral symptoms. The mind in some cases remains perfectly clear; in others there is delirium, but seldom continuous, deep coma.

At its beginning, cerebro-spinal meningitis may be confounded with pneumonia or other diseases with cerebral symptoms. It is differentiated with certainty only by lumbar puncture. It is sometimes difficult to distinguish between cerebro-spinal and tuberculous meningitis. The former is relatively infrequent except in epidemics. The fluid in cerebro-spinal meningitis is usually turbid and contains many cells of the polymorphonuclear variety; in tuberculous meningitis the fluid is generally clear and the few cells found are lymphocytes. Tuberculous meningitis may occur anywhere or at any time. Its characteristics are a gradual onset with indefinite symptoms, low temperature, drowsiness, irregularity of pulse and respiration, absence of active delirium, late coma, less marked hyperæsthesia and rigidity, duration seldom over three weeks from the beginning of definite cerebral symptoms, termination invariably fatal. Cerebro-spinal meningitis, however, frequently ends in recovery, and it is the only form of acute meningitis which does so.

**Treatment.**—Flexner of the Rockefeller Institute has developed a serum for the treatment of cerebro-spinal meningitis which has been shown to be more effective in controlling the disease than any other measure thus far proposed. The serum is obtained by immunising horses with toxins and cultures obtained from many strains of the meningococcus. It acts chiefly on the bacteria themselves, and only to a slight degree on their products; i. e., it is a bacteriolytic serum. It is used as follows: After withdrawing by lumbar puncture all the fluid that will flow readily, under the strictest aseptic precautions, the serum, warmed to the body temperature, is injected without removing the needle. In some exceedingly sensitive patients the administration of a few whiffs of chloroform may be necessary. The injection should be made very slowly, occupying several minutes. Raising the hips facilitates the inflow of the serum. To be effective, it must be brought into contact with the organisms in the spinal canal in a considerable degree of concentration.

The initial dose is 30 to 40 c.cm., which should be repeated in twelve hours if there is no improvement in the symptoms. Usually the second dose is not given until the end of twenty-four hours, and after that a daily dose of the same size for four or five days should be given, unless there is a prompt disappearance of all symptoms. Injections should be continued so long as organisms are found in the fluid or nervous symptoms, fever, and leucocytosis persist. If done cautiously, it is safe to introduce more serum than the amount of fluid withdrawn. In the milder cases it sometimes happens that a single dose may suffice for a cure; but even under such circumstances it is safer to give at least three doses. The serum arrests the inflammatory process by destroying the organisms which produce it. To accomplish this a sufficient dose must be given, and given early, before important inflammatory changes have taken place.

An immediate effect of the injection is seen in the cerebro-spinal fluid. There is a marked reduction in the percentage of polymorphonuclear cells. The number of meningococci is greatly reduced and their vitality lessened. After the first injection they stain with difficulty, and after a second injection it is generally impossible to grow them, although they are usually present in small numbers (Fig. 115). The effect on the symptoms is striking. There is a marked reduction in the temperature, which may amount to three or four degrees in twenty-four hours, and it may not rise again (Fig. 116). The stupor and delirium often diminish rapidly, and soon disappear. Improvement is also seen in the patient's general condition, pulse, and respiration. The last symptoms to be affected are usually the rigidity of the neck and extremities.

The results of this treatment show a much larger percentage of recoveries than has been obtained by any other method.<sup>1</sup> Of 1,500 cases of all types, in patients of all ages, thus far treated by this serum, the general mortality was about 25 per cent. The figures represent results obtained in many epidemics in all parts of the world. The statistics from this country are not so favourable as those from abroad with the same serum, for the reason that in the results here are included reports from many physicians who, without experience in the use of the serum, treated but one or two cases. The foreign statistics, however, are in larger groups, and the cases for the most part were under the care of men who had had experience with the serum. In the recent epidemic in France the mortality of the cases not treated by serum was about 70 per cent, while in those receiving serum it was but 15 per cent. This indicates what may be expected with serum treatment under favourable conditions. One of the most striking evidences of the value of this treatment is the results obtained in infants under one year. Without serum these cases

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<sup>1</sup> For details, see articles by Flexner and his associates in the *Journal of Experimental Medicine*, from September, 1908, to 1911. The serum can be obtained from the New York Health Department.



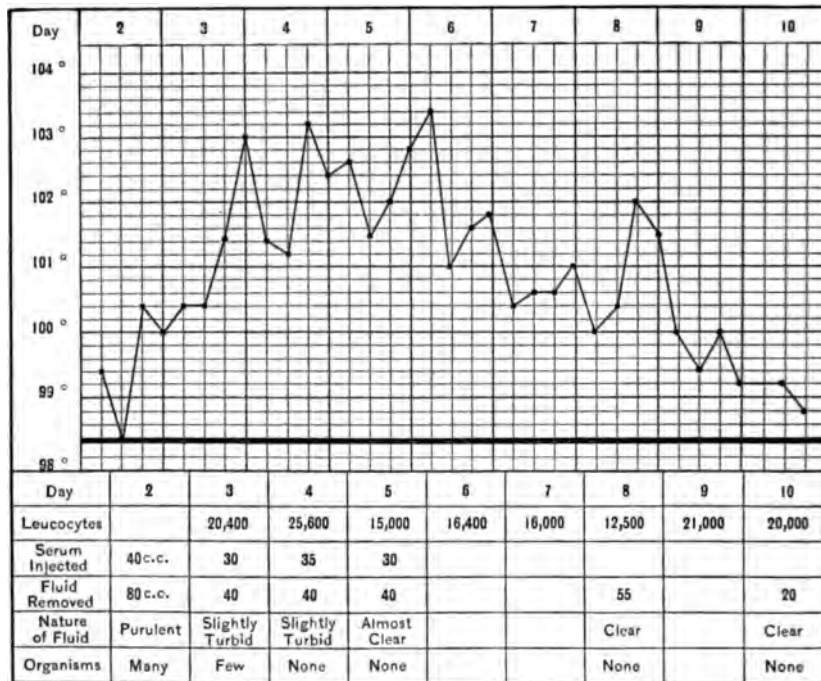


FIG. 115.—CEREBRO-SPINAL MENINGITIS TREATED BY SERUM. Infant, 7 months old, Babies' Hospital: 24 hours ill; intense prostration; respiration, 80; signs of pulmonary oedema; general relaxation; stupor; profuse hæmorrhagic eruption. First fluid, purulent; amount removed, amount of serum injected, and the changes in the fluid shown in the chart. Immediate improvement in symptoms after first injection. Subsequent symptoms typical. A rise in temperature on the 8th day and the increase in leucocytes on the 9th and 10th days suggested relapse; but as fluid was clear and no organisms could be found in smears or by culture no more serum was given; complete recovery.

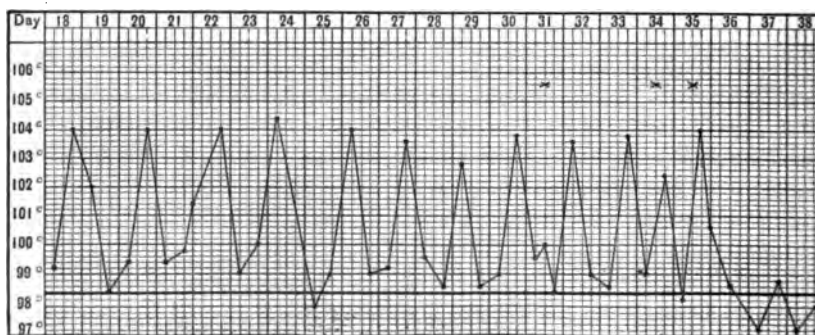


FIG. 116.—CEREBRO-SPINAL MENINGITIS. Late injection of the serum, prompt effect; complete recovery. Boy, 11 years, St. Vincent's Hospital, New York. Early symptoms obscure, and on account of swelling and pain in joints diagnosis of rheumatism made; cerebral symptoms not marked. First lumbar puncture made on 31st day and meningococcus found. Serum injected on the 34th and 35th days. Rapid fall in the temperature followed by cessation of all symptoms and complete recovery.

have almost invariably terminated fatally; with serum over 50 per cent of them have recovered.

The results are much modified by the time of injection as shown by the following table:

*Mortality of serum-treated cases according to time of injection.*

| TIME OF INJECTION.          | Flexner.<br>(All sources,<br>chiefly U. S.) | Netter.<br>(France.) | Dopter.<br>(France.) |
|-----------------------------|---|----------------------|----------------------|
| 1st to 3d day . . . . .     | 14.9%                                       | 7.14%                | 8.20%                |
| 4th to 7th day . . . . .    | 22.0%                                       | 11.1%                | 14.4%                |
| After the 7th day . . . . . | 36.4%                                       | 23.5%                | 24.1%                |

In Netter's series Flexner's serum was used; Dopter used the serum prepared at the Pasteur Institute.

The effect on the course and duration of the disease is no less marked than that upon the mortality. The duration of acute symptoms is very much shortened, and in about one-fourth of the cases the disease terminated by crisis (Figs. 116, 117). This was more often seen in cases injected early, although it was observed in some injected as late as the fourth week. The infrequency of complications and sequelæ is also noteworthy. Not only do patients recover, but they recover quickly, and in most instances completely. The absence of complications and sequelæ is, no doubt, to be explained partly by the effect of the serum in shortening the disease.

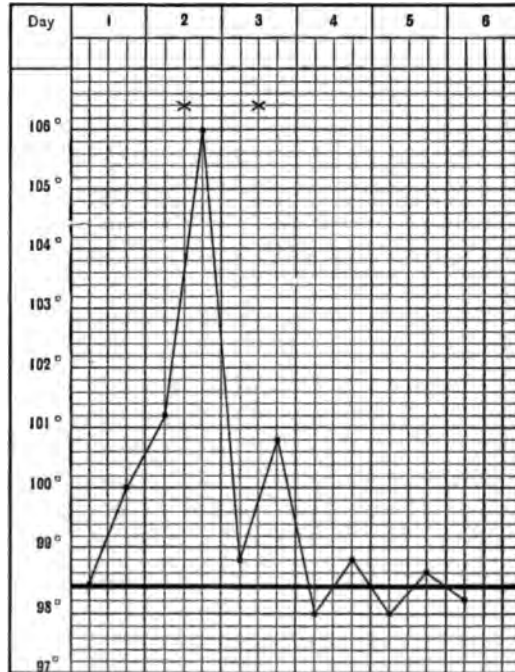


FIG. 117.—CEREBRO-SPINAL MENINGITIS. Termination by crisis; recovery after two injections. Boy of 6 years, patient of Dr. C. H. Dunn. The first day, rather indefinite symptoms—headache, vague pains, slight fever. Second day, alarming symptoms rapidly developed and patient became comatose; 30 c.cm. of serum given and repeated the following day. The temperature rapidly fell and did not again go above 101° F. In twelve hours the coma was gone and the mind clear; by the fourth day the child was convalescent. No subsequent symptoms.



The nutrition of the patient is important. Feeding is often difficult, and gavage may be advantageously employed. Bed-sores should be prevented by cleanliness, frequently changing the patient's position, etc. Retention of urine may require the use of the catheter.

For the residual paralysis, massage, warm baths, and friction should be employed, but electricity only when all symptoms of central irritation have subsided. The prolonged use of iodide of potassium, especially in combination with mercury, seems to have some value.

#### ACUTE MENINGITIS DUE TO OTHER CAUSES.

Besides the main varieties of acute meningitis, viz., that due to the meningococcus and that due to the tubercle bacillus, there are other forms differing in etiology, but closely related clinically, and therefore they may be advantageously considered together. It is only since the general adoption of lumbar puncture as a means of diagnosis that these forms of meningitis have been clinically differentiated. Formerly they were grouped under the somewhat indefinite heading of "simple meningitis." Three of these varieties, those due to the pneumococcus, the influenza bacillus, and pyogenic organisms, are sufficiently important to require separate description. Cases of meningitis due to the typhoid bacillus, the gonococcus, and the colon bacillus, have all been reported in children, but are so rare as only to deserve mention.

**Pneumococcus Meningitis.**—This is the most important variety included in this group and the one most frequently met with in young children. In my hospital patients eleven per cent of the cases of acute meningitis were of this form. All had pulmonary symptoms of greater or less severity, and two-thirds of the patients had definite pneumonia with consolidation; several had also empyema. Less frequently, pneumococcus pericarditis and peritonitis have been present. Occasionally pneumococcus meningitis is seen when there are no definite pulmonary symptoms or signs and when it is apparently a primary inflammation. However, in most cases pneumococcus meningitis is one of the results of a generalised pneumococcus infection. In every one of seven cases of pneumococcus meningitis of my own in which cultures of the heart's blood were made at autopsy, this organism was present. This form of meningitis occurs in infants more frequently than in older children, and, in my experience, usually in very young infants; over half of the cases seen were in patients under six months old. While the disease usually develops at the height of an attack of pneumonia, it may precede the pulmonary symptoms and it may develop during convalescence. I once saw it as late as the fourth week.

**Lesions.**—In a general way the anatomical changes resemble those described in cerebro-spinal meningitis, with the exception that the

marked changes in the brain substance which are usually dependent upon the long course of that disease are wanting. As a rule, also, the lesions are limited to the brain. If the cord is involved, it is only to a slight degree.

Acute meningitis due to the pneumococcus is characterised by a more abundant exudation of fibrin and pus than is seen in any other variety of meningitis. The lesion may affect the entire brain, but it is especially marked at the convexity and over the anterior lobes. Sometimes it is limited to these regions, the meninges of the base escaping. The exudate may be so abundant as almost to conceal the convolutions. (See Plate XIV.) There is usually less distention of the ventricles than in cerebro-spinal meningitis.

In cases apparently primary, or when meningitis occurs very early in the course of a general pneumococcus infection, the symptoms are usually indistinguishable from ordinary cases of cerebro-spinal meningitis. It is generally not until lumbar puncture is made that the variety of meningitis is suspected. When meningitis occurs as a secondary inflammation it is often latent, and not infrequently is found at autopsy when not suspected during life. Usually, however, the meningeal complication is indicated by the abrupt development, in the course of an attack of pneumonia, of vomiting or convulsions, followed by active delirium or stupor. Because the lesion is principally, sometimes only, at the convexity, many of the symptoms belonging to meningitis with basal lesions are absent. There is rarely cervical opisthotonus; the fontanel may not be bulging; pulse and respiration may not be disturbed; in fact, there are no cranial nerve symptoms and the symptoms due to spinal involvement—hyperæsthesia, rigidity, Kernig's sign, etc., are usually wanting.

The course of pneumococcus meningitis is generally short and acute, death taking place within three or four days from the first symptoms. I have twice seen a prolonged type of the disease lasting many weeks; one case ended fatally near the end of the third month; the other patient recovered from the acute symptoms, but remained partially paralysed and mentally defective.

The diagnosis of pneumococcus meningitis can positively be made only by lumbar puncture. The cerebro-spinal fluid in gross appearance does not differ from that seen in cases due to the meningococcus. The cells present are chiefly polymorphonuclear. Pneumococci are very abundant and are easily found in smears and grown readily in cultures. The existence of pneumococcus meningitis is not always shown by lumbar puncture. I have met with one case in which repeated punctures gave negative results, and yet the autopsy showed meningitis to be present, but only the convexity was affected. The organisms were readily found in the meningeal exudate.

PLATE XIV.



**ACUTE MENINGITIS, COMPLICATING PLEURO-PNEUMONIA.**

Child twenty months old; on twenty-third day of a protracted attack of pneumonia, vomited six times, and the temperature, which had been nearly normal for four days, rose to 103° F. On the following day general convulsions, which were repeated frequently during the next few days; temperature, 101° to 104° F.; death in convulsions on twenty-eighth day.

*Autopsy.*—Pleuro-pneumonia of left side; lung resolving. Anterior portion of brain enveloped in lymph and pus, more marked at the convexity, but present also over the base.



**Influenza Meningitis.**—This form of meningitis is rare, but in many respects resembles the form just described. According to Wollstein,<sup>1</sup> there had been recorded, up to 1911, 49 cases of pure, and 9 cases of mixed, influenza meningitis. Of these, 28 were in infants under one year old. Of the reported cases, 5 recovered, 2 of these being in infants. Of the 5 cases which have come under my own observation, one was in a boy of four years; the others were in infants. All ended fatally. In my experience, influenza meningitis has been secondary to other influenza infections, usually those of the naso-pharynx or bronchi. The organisms were found by culture from the secretions of these parts during life. One patient, an infant of eight months, was admitted to the hospital with an acute abscess of the elbow. Two days later symptoms of meningitis developed, and death occurred in three days. The autopsy showed an extensive purulent meningitis. Pure cultures of the influenza bacillus were obtained from the pus of the elbow, the fluid drawn by lumbar puncture, the meningeal exudate, and the heart's blood. The lungs showed influenza bacilli and streptococci.

The lesions of influenza meningitis, in the few cases in which autopsies have been made, have differed in no essential particular from those described in the pneumococcus variety. In three of the cases coming under my observation in which examinations were made, the influenza bacillus was obtained from the heart's blood as well as from the cerebro-spinal fluid.

Clinically, influenza meningitis runs a short, very acute course. There are no features by which it can be distinguished from the pneumococcus or meningococcus form, except the findings of lumbar puncture. In gross appearance the fluid does not differ from that seen in the other forms. There is usually marked turbidity; the cells are abundant and of the polymorphonuclear variety. The organisms are generally not numerous in the smears, in marked contrast to the other forms of meningitis. They are readily grown upon blood agar, but not upon ordinary media. If, therefore, from a turbid cerebro-spinal fluid no growth occurs, influenza meningitis should be suspected.

**Meningitis Due to Pyogenic Organisms—Septic Meningitis.**—Meningeal inflammations set up by the streptococcus and staphylococcus are not very common in young children. They are almost always secondary. In the newly born this form of meningitis is seen in general pyæmia, usually from umbilical infection; it also follows infection of a spina bifida. In older children it follows injuries to the head, erysipelas of the scalp, operations upon the brain, and otitis media with mastoiditis or sinus thrombosis. Such a complication of otitis in infancy is, however, extremely rare. The lesions consist in a widespread general in-

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<sup>1</sup> American Journal of Diseases of Children, January, 1911.



flammation of the pia with an abundant exudate of pus, but with less fibrin than in the two varieties previously described.

The symptoms of septic meningitis are not distinctive. The course is usually a rapidly progressing one, and the termination almost invariably in death. The fluid drawn by lumbar puncture in most cases is markedly turbid, and shows great numbers of pus cells. The organisms are present in large numbers and are readily recognised both in smears and by cultures upon ordinary media.

**Diagnosis.**—The differential diagnosis of the different forms of meningitis from each other, and from other diseases with cerebral symptoms, is made with certainty only by means of lumbar puncture, which should be done in all cases of doubt. The appearance of the cerebro-spinal fluid is essentially the same whether the inflammation is due to the meningococcus, the pneumococcus, the influenza bacillus, or to the staphylococcus or streptococcus. The symptoms of meningitis in general, fully described in the chapter on Cerebro-Spinal Meningitis, are present in most of the cases.

**Prognosis and Treatment.**—The prognosis in all varieties of acute meningitis, except that due to the meningococcus, is very bad; almost every case of meningitis due to other causes is fatal. From what has been said, it would appear that treatment is as yet most unsatisfactory, and only symptomatic. Wollstein's researches at the Rockefeller Institute, however, indicate that influenza meningitis may yet be controlled by serum treatment. A goat serum has been produced which regularly controls the experimental disease in monkeys, although its use has not yet been extended to man.

### TUBERCULOUS MENINGITIS.

(*Acute Hydrocephalus; Basilar Meningitis.*)

Tuberculous meningitis is a tuberculous inflammation of the pia mater of the brain, sometimes involving also that of the cord. It is by far the most frequent form of acute meningitis seen in young children. In my hospital experience, apart from epidemics of cerebro-spinal meningitis, seventy per cent of the cases of acute meningitis have been tuberculous. It is more uniformly fatal than any other disease of early life. It is doubtful if it ever occurs as the only tuberculous lesion of the body. In infancy it is usually associated with general or pulmonary tuberculosis; in older children with tuberculosis of the bones, joints, or lymph nodes. Of my own cases, forty per cent of all deaths from tuberculosis in children have been due to meningitis.

**Lesions.**—The lesion consists in the production of miliary tubercles, with which are frequently found tuberculous nodules of variable size, and in almost every case there are also the products of ordinary inflammation

of the pia mater—fibrin and pus—together with an accumulation of fluid in the lateral ventricles of the brain. Frequently there are tubercles in the pia mater of the upper portion of the cord. When few in number the tubercles are usually only at the base. When numerous they are seen scattered over the convexity. Tubercles are sometimes found in the choroid coat of the eye. The amount of fibrin and pus in the exudate is usually small, and is much less than is seen in other forms of acute meningitis. The inflammatory products are most abundant at the base. In addition to the patches of greenish-yellow fibrin, there are adhesions between the lobes of the brain and thickening of the pia. In cases which have lasted for several weeks, this thickening may be marked, owing to cell infiltration and the production of new connective tissue. The pia is studded with miliary tubercles, sometimes with small yellow tuberculous nodules; frequently there is arteritis, which is sometimes obliterating.

In the most acute cases the brain substance immediately beneath the pia is intensely congested, slightly softened, and shows under the microscope a superficial encephalitis. The lateral ventricles are usually distended with clear serum, sometimes with serum containing flocculi of fibrin or pus: the amount present varies from one to four ounces in each ventricle, being always greater in the subacute cases. The walls of the ventricles may be softened. The distention of the ventricles leads to flattening of the convolutions from pressure against the skull, to bulging of the fontanel, and sometimes to separation of the sutures.

Tuberculous nodules varying in size from a small pea to a walnut are frequently seen associated with meningitis in older children, but not often in infants. These nodules may be connected with the meninges, or they may be situated within the brain substance, usually in the cerebellum. The larger ones are classed as brain tumours. Inflammatory products are rarely found in the spinal canal.

Although it is not infrequent to see meningitis without symptoms of tuberculosis elsewhere, I have never failed at autopsy to find other tuberculous lesions in the body. In my own experience the following are those most often met with, given in the order of frequency: (1) In infants, associated with general or pulmonary tuberculosis; (2) in children from three to twelve years of age, with tuberculosis of the vertebræ, hip, knee, or ankle; (3) at any age, with tuberculosis involving only the tracheal, bronchial, or mesenteric lymph nodes; (4) much less frequently with the pulmonary tuberculosis of older children.

**Etiology.**—Tuberculous meningitis is produced only by the transportation of the tubercle bacilli to the brain. They may find their way by the blood-vessels or lymphatics.

The following table shows the age at which the disease was observed in 410 cases of which I have notes:

|                             |     |
|-----------------------------|-----|
| Under one year .....        | 162 |
| One to two years .....      | 149 |
| Two to five years .....     | 76  |
| Five to nine years .....    | 17  |
| Nine to sixteen years ..... | 6   |
| Total .....                 | 410 |

In this series three cases were in children three months old or younger. Tuberculous meningitis occurs much more often in the winter

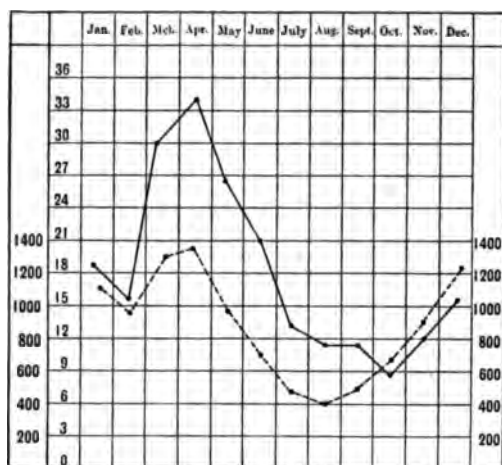


FIG. 118.—SEASONAL OCCURRENCE OF TUBERCULOUS MENINGITIS. The upper curve (—) represents the seasonal occurrence of 218 cases of tuberculous meningitis. The lower curve (---) represents the deaths from pneumonia in New York City for one year.

and spring months than at other seasons (Fig. 118). The most plausible explanation of this seems to be that these patients, infected some time previously, carry a latent focus of tuberculosis somewhere in the respiratory tract, usually the bronchial glands. Under the influence of acute respiratory infections of the cold season, the latent tuberculous disease becomes active, and a rapidly spreading tuberculous process results. In infants and young children it rarely happens that pulmonary

lesions are absent; but these patients are especially predisposed to early meningeal infection, and this often occurs before symptoms of tuberculosis elsewhere have manifested themselves. At the time of invasion, therefore, most of these children are apparently in the best of health. In older children there may have been previous evidence of tuberculosis in lungs, bones, or lymph nodes. The modes of acquiring tuberculosis are discussed in the general chapter on that disease. It is sufficient to say here that it is usually from some member of the family or household. This may be not only a person who is in the active stage of pulmonary tuberculosis, but one who is supposed to be cured or one in whom the disease has not yet been suspected. Exposure may antedate symptoms by several weeks or months. Striking evidence in favor of the human origin of tuberculous meningitis is obtained from a study of the type of tubercle bacillus present in cases of meningitis. In thirty-two cases in my series, this was worked out by Dr. W. H. Park in the Research

Laboratory of the New York Health Department. In thirty the bacillus was of the human type; in one it was of the bovine type, and in one both types were present.

**Symptoms.**—In about two-thirds of the cases the onset is gradual; but in a considerable number of those classed as abrupt, careful inquiry will elicit a history of previous indisposition. The most frequent early nervous symptoms are: disinclination to play, drowsiness, or sometimes constant fretfulness or irritability. Often there is a complete change in disposition. In a case under my observation this was most striking; a little girl previously devoted to her mother, could not endure her presence in the room. Sleep is restless and disturbed; there may be grinding of the teeth. Older children often complain of headache. At all ages, but particularly in infancy, early digestive symptoms are prominent. There are seen frequent attacks of vomiting without apparent cause; the bowels are generally constipated and the appetite is almost entirely lost. Usually there is also a slight but continuous elevation of temperature. Indefinite symptoms may last for four or five days, or they may be spread over two or three weeks without perhaps being sufficiently severe to attract much notice. Finally, unmistakable evidence of brain disease develops. The early disturbances are often ascribed to dentition, or to indigestion.

In most cases the first pronounced cerebral symptom is persistent and increasing drowsiness; exceptionally it is an attack of general convulsions, followed in a few hours by stupor. Often a period of irritative symptoms is present, lasting several days. There is headache, usually located in the frontal region, and occasionally photophobia; sometimes pain is indicated by the child's suddenly screaming out at night, which may be repeated many times without waking; sometimes during the greater part of the time for two or three days these frequent screaming attacks may be repeated. The skin is somewhat hyperæsthetic; the reflexes are apt to be exaggerated; the muscles of the neck may be rigid and the head is drawn back, or there may be rigidity of the extremities. The pupils are normal or contracted; there may be nystagmus. The child is fretful, wishes to be left alone, and cries if disturbed. In some cases these symptoms are so marked as strongly to suggest cerebro-spinal meningitis. They may alternate with periods of marked apathy and dulness. During this stage there is occasional vomiting, and the bowels are obstinately constipated. The pulse is usually somewhat accelerated, but may be slow and occasionally it is irregular. The respiration is of normal frequency, but a careful observation during sleep or perfect quiet will often show a slight irregularity which is very significant. The temperature is usually elevated, ranging from 99° to 100.5° F. When a high temperature is seen, it is usually due to tuberculosis elsewhere than in the brain.

As the disease advances, the irritative symptoms subside, and the stupor becomes deeper and more continuous. If undisturbed, the child may sleep a great part of the time, but can be roused, and then appears quite rational. Finally the stupor becomes so profound that the child can not be roused at all. Active delirium is rare. The pupils respond slowly to light or not at all; they may be unequal; occasionally there is seen strabismus, ptosis, or paralysis of the face. More often there is hemiplegia, or paralysis of one arm or leg. Such paralyzes are often transient, disappearing after a day or two. Automatic movements of the extremities, particularly of the arms, are frequent. Muscular twitchings may be noticed. Opisthotonus is marked and well-nigh constant. In infants the fontanel is tense and bulging. In older children especially the abdomen is retracted, giving the typical "boat-belly." On drawing the finger-nail along the skin of the abdomen, there appears a distinct red streak, which remains for several minutes. This is the *tâche cérébrale*, and it is almost always present. Other vaso-motor disturbances may be seen. The reflexes are variable; in the early part of the disease they are usually increased, later they are diminished or abolished. The pulse now becomes slow and irregular, often intermittent. The respiration is almost always irregular; a very characteristic type consists in the movements becoming deeper and deeper until there is a sigh; after a complete arrest of respiration for several seconds the phenomenon is repeated.

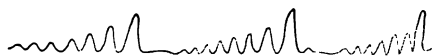


FIG. 119.—TRACING OF RESPIRATION IN TUBERCULOUS MENINGITIS.

The accompanying tracing illustrates the type (Fig. 119). An examination with the ophthalmoscope usually shows the presence of choked discs.

The progress of the disease is subject to great variations, especially in children over two years old. The advance of symptoms is slower and is interrupted by periods of remission which may continue two or three days. After being in quite deep stupor, a child may recover consciousness, and even sit up and play with toys, leading to the view that an error in diagnosis has been made. But this respite is only temporary; soon the child passes again into coma.

From this time the duration of the disease is from three to ten days. The child can not be roused at all. The pupils are widely dilated, and do not respond to light. There is general muscular relaxation. There may be retention of the urine. Deglutition is difficult, often impossible. The respiration is more rapid, but still irregular. The pulse becomes very rapid and feeble, often 160 to 180 a minute. Toward the end the temperature rises rapidly to 101° F., sometimes to 106° or 107° F. (Fig. 120). Death usually takes place from exhaustion in deep coma, or convulsions develop and continue from twelve to twenty-four hours until death. Sometimes a patient will live for days in a condition of

prostration so extreme that death is hourly expected. A rapidly rising temperature or the occurrence of late convulsions usually indicates approaching death. Of fifty-seven cases, fifty died in coma, seven in con-

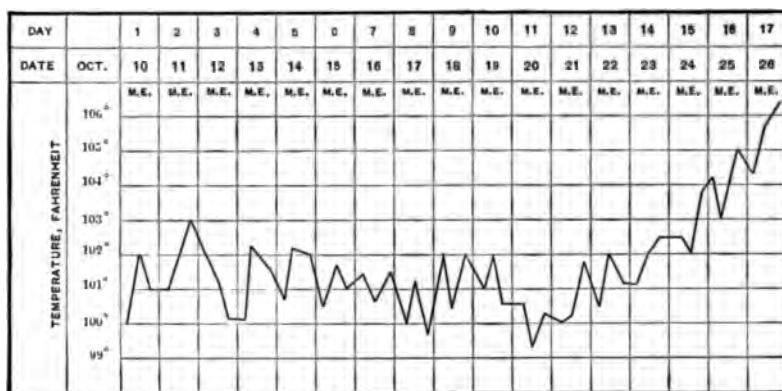


FIG. 120.—FAIRLY TYPICAL TEMPERATURE CURVE IN TUBERCULOUS MENINGITIS. Boy, twenty months old; death on seventeenth day.

vulsions. The entire duration of the disease from the beginning of definite nervous symptoms is rarely over three weeks, and in infants it is usually shorter than this.

**Diagnosis.**—Tuberculous meningitis is often overlooked because the patients do not give outward evidences of tuberculosis. Its great frequency should always lead one to suspect it when protracted nervous symptoms are present in infants. There are no diagnostic symptoms in the early stage. The indefinite symptoms that belong to this stage of the disease are frequent in young children suffering from chronic indigestion associated with constipation. Cases of cyclic vomiting may present many of the symptoms of meningitis.

The most diagnostic symptoms of tuberculous meningitis enumerated in the order of their frequency are as follows: persistent drowsiness, obstinate constipation, vomiting without apparent cause, irregular respiration, irregular pulse, convulsions, opisthotonus, and fever which is usually slight. A positive diagnosis is made only by lumbar puncture; by this means this form is distinguished from other forms of acute meningitis.

The fluid drawn by lumbar puncture is usually perfectly clear, but sometimes after standing there is a slight deposit present. Exceptionally the fluid may be turbid. The cells are usually few in number, and of the mononuclear variety. The presence or absence of sugar has been in my experience of no diagnostic importance.

Tubercle bacilli are, I believe, invariably present in the fluid, and by careful examination can be found microscopically in nearly every case. They were found in 135 of 137 consecutive cases of tuberculous

meningitis at the Babies' Hospital.<sup>1</sup> They are more numerous late in the disease.

The technique is important. Fluid should be drawn into several tubes and the last one containing 15 or 20 c.cm. set aside for examination, as the bacilli are much more likely to be found in this than in the first fluid. The tube should not be shaken, but should be allowed to stand for twelve hours, preferably in an incubator. A central coagulum or film generally forms in the fluid, and in this the bacilli are usually entangled. This should be spread out entire and carefully examined. In other cases the bacilli may be found after centrifuging; in still others by scraping the sides of the tube with a platinum loop or by examining superimposed drops which have been allowed to dry upon a slide. In most of the cases the number of bacilli present is not large and the average length of search required has been about an hour, but in a few instances the number is so large that they are present in practically every field.

Noguchi's globulin test<sup>2</sup> is useful in distinguishing inflammatory from normal cerebro-spinal fluids. It is, however, of no value in distinguishing between the different forms of meningitis. A positive reaction is obtained with great uniformity in every variety of acute meningitis. This test is of special assistance in the tuberculous cases, for in them the gross appearance of the fluid does not usually differ from the normal; moreover, it gives early information.

Bacilli have been found in the sputum, in my experience, in nearly one-half the cases in infants and young children, although in most of them there was either no evidence of pulmonary disease, or only cough and a few scattered râles in the chest.

The v. Pirquet cutaneous test gives reliable information except in moribund cases, in those excessively prostrated or with very poor circulation. A positive reaction was obtained in 51 of 65 consecutive cases tested, the negative results being usually for the reasons mentioned. This test is of much assistance in early diagnosis. If then a

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<sup>1</sup> See Hemenway, American Journal of Diseases of Children, January, 1911.

<sup>2</sup> The test is as follows:

To 0.5 c.cm. of a 10-per-cent solution of butyric acid in 0.9-per-cent salt solution add 0.1-0.2 c.cm. of suspected fluid and boil.

Then add 0.1 c.cm. normal sodium hydrate solution and boil again.

No change in the solution or only a faint cloudiness is to be considered a negative reaction.

A flaky precipitate is a positive reaction.

NOTE.—The test should be controlled by boiling the two solutions without the suspected fluid. The reagents should be freshly made; they may change after two or three weeks. The accidental addition to the fluid of even a few drops of blood spoils the test, for this is sufficient to give the globulin reaction although no meningitis is present.

child with symptoms distinctly meningeal gives a positive reaction to the tuberculin test the probabilities of tuberculous meningitis are greatly strengthened, even though at the time bacilli may not have been found in the cerebro-spinal fluid.

The cerebral symptoms of intestinal and many other acute diseases sometimes closely resemble those of tuberculous meningitis. From all such the diagnosis is made by lumbar puncture. In any case of meningitis in a young child the chances are greatly in favour of the tuberculous form, since it is much more frequent. The diagnosis of tuberculous meningitis from the cerebral form of acute poliomyelitis is at times difficult. It is discussed under the latter disease.

**Prognosis.**—Although there have been recorded a few instances of recovery after the tubercle bacilli have been found in the fluid obtained by lumbar puncture, such an outcome is not to be expected. I have never seen such a case recover. The reported recoveries in which the diagnosis has rested upon clinical symptoms only can not be accepted.

**Treatment.**—From what has been said regarding prognosis, it follows that if the diagnosis is correct the case is practically hopeless, no matter what treatment is employed; but as a positive diagnosis is not always possible, all cases should be treated like other forms of acute meningitis.

#### CHRONIC BASILAR MENINGITIS IN INFANTS.

It was first pointed out in 1898 by Still that this disease is usually due to the *diplococcus intracellularis*; in other words, that it is a chronic form of cerebro-spinal meningitis. Chronic basilar meningitis is most frequently seen after epidemics of cerebro-spinal meningitis, but it is occasionally met with at other times as a sequel of a sporadic case. It occurs after an acute attack, when the basilar lesion persists and becomes chronic. As acute cerebro-spinal meningitis in infants is usually fatal if the attack is severe, it follows that the chronic form is seen only after the mild attacks. It is chiefly for this reason that the early symptoms often are not recognised as those of cerebro-spinal meningitis. The patient frequently does not come under observation until all acute symptoms have passed away, the persistent opisthotonus being the chief feature of the case.

There is also seen in children, though very rarely, a chronic basilar meningitis of syphilitic origin. At least two such cases have come under my observation in the Babies' Hospital. One was cured by anti-syphilitic treatment, and in the other the diagnosis was confirmed by autopsy.

**Lesions.**—This process is usually limited to the base of the brain. The pia mater is thickened about the interpeduncular space, also over the medulla, pons, and cerebellum. These different parts may be adherent to each other, or to the inner surface of the dura. The cranial nerves may



be compressed. The openings in the fourth ventricle are usually obliterated, and there results a distention of the lateral ventricles with clear serum, sometimes in sufficient amount to be regarded as hydrocephalus. Rarely, pus may be found in the ventricles.

**Symptoms.**—The onset is usually gradual, although in most cases there can be obtained a fairly distinct history of an early active period. The most prominent symptoms are cervical opisthotonus, moderate hydrocephalus, and usually general muscular rigidity. The opisthotonus is often extreme (Fig. 121) and is greater than is seen in any other disease. If placed upon its back the body of the child often touches the table only at the occiput and the sacrum (Fig. 122). The head is usually somewhat enlarged, but never to the degree seen in primary hydrocephalus; the fontanel bulges, and the sutures are separated. These symptoms are due to an accumulation of fluid in the lateral ventricles; they are never so marked as in primary hydrocephalus. The rigidity of the extremities is very great and in most cases constant; the legs and feet



FIG. 121.—CHRONIC BASILAR MENINGITIS—EXTREME DEFORMITY. Ill for five months; followed cerebro-spinal meningitis; posture shown in the picture maintained for the last six weeks; death at ten months. Autopsy showed typical lesions.

are usually extended, while the forearms are flexed and the hands clenched. All the reflexes are greatly exaggerated. There is rarely coma, but mental dulness alternating with periods of great irritability in which general convulsions may occur. Vision may be impaired or wanting entirely. The fact that in most cases optic neuritis is absent is of some value in differentiating this disease from tumour. Nystagmus is often present and attacks of vomiting occur without evident cause. There is no fever except for a few days at a time during acute exacerbations. The usual duration of the disease is from two to five months;

death may occur from convulsions, or from some intercurrent disease, such as pneumonia, but most frequently from marasmus. The prognosis is very bad except when the cause is syphilis, when recovery may take place.

**Diagnosis.**—The disease is to be distinguished from tuberculous meningitis, and from the opisthotonus of reflex origin which is occasionally seen in infants suffering from marasmus. It differs from tuberculous

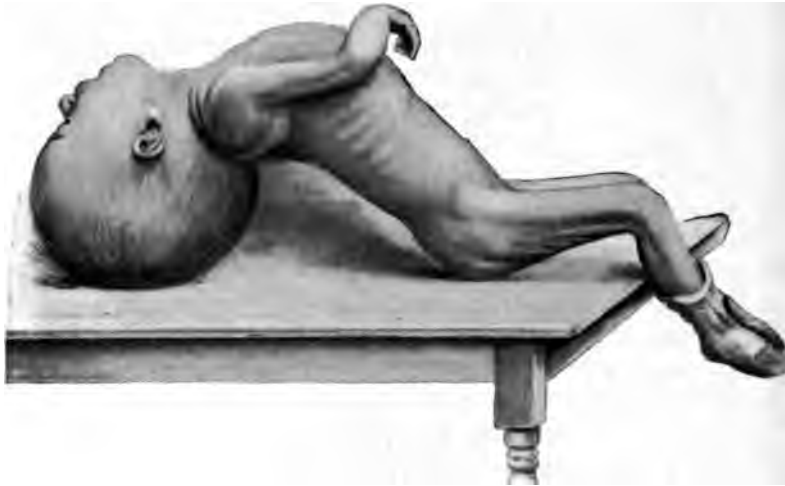


FIG. 122.—CHRONIC BASILAR MENINGITIS.  
A patient in the Babies' Hospital (diagnosis confirmed by autopsy).

meningitis in its more protracted course, in the absence of fever and paralysis, and also in the greater prominence of the opisthotonus and hydrocephalus.

**Treatment.**—If there is any reason to suspect syphilis, salvarsan and the iodide of potassium and mercury should be administered. Lumbar puncture is useful for diagnosis only. The establishment of auto-drainage of the ventricles, as practised in primary hydrocephalus, has recently been advocated for this condition, and tried with some measure of success.

#### THROMBOSIS OF THE SINUSES OF THE DURA MATER.

This is not very frequent. It may depend upon certain general conditions, when it is usually classed as *cachectic* or *marantic thrombosis*; it may be associated with local pathological processes, when it is known as *inflammatory* or *septic thrombosis*.

**Cachectic Thrombosis.**—This is seen in infants and young children, but is very rare after the age of five years. It occurs in the course of various diseases, the most frequent being pneumonia, pertussis, diphtheria, nephritis, tuberculosis, and the acute intestinal diseases. In

connection with the last-mentioned group, altogether too much has been made of it, as it is really rare, and in only a very few cases does it explain the cerebral symptoms present. The actual cause of the thrombosis is the altered condition of the blood and the feeble circulation, as the walls of the sinuses are normal.

The most frequent seat of cachectic thrombosis is the superior longitudinal sinus. At autopsy one must be careful not to confound the soft, partly decolourised, non-adherent thrombi of post-mortem origin, with those of ante-mortem formation. The latter are firm, and when of long standing may be very hard and even show a laminated structure. They usually fill the sinus completely, and are adherent. The thrombus extends from the sinuses to the veins emptying into it, which stand out like dark worms upon the surface of the brain. The brain itself may be deeply congested, or it may be covered with a diffuse hæmorrhage, but more frequently the brain and the membranes are simply œdematous.

The symptoms of cachectic thrombosis are few and uncertain, and in a large number of cases the disease is latent. Very rarely is a positive diagnosis possible during life. When the thrombosis occurs just before death, its symptoms are so mingled with those of the original disease that they can not be separated. In some cases there may be localised or general convulsions, or paralysis, loss of consciousness, and strabismus.

The prognosis is bad, cases generally proving fatal in the course of a few days. The diagnosis is so uncertain and obscure that the treatment must be symptomatic, and directed toward the general rather than the local condition.

**Inflammatory Thrombosis—Septic Thrombosis—Sinus-Phlebitis.**—This condition is most frequently seen in children in connection with acute meningitis. It may exist either with the simple or the tuberculous variety. It also follows otitis—especially old and neglected cases—usually with necrosis of the petrous bone, but sometimes without it. It is much less frequently associated with disease of the ear in children than in adults. It may arise from traumatism, necrosis of the cranial bones, or from septic processes involving any of the cavities or any of the structures adjacent to the brain, such as the scalp, orbit, nasal fossa, mouth, or pharynx. Infection from the mouth or pharynx is most frequent in children in connection with scarlet fever or diphtheria; while usually secondary to otitis it may occur without it, the infection being carried by the blood-vessels. Infection from the nose may have its origin in ulceration from syphilis or tuberculosis. In the orbit, the source may be malignant disease.

The seat of the thrombosis will depend upon the original disease. If this affects the cranial bones or the scalp, it will be the longitudinal sinus; if the ear, the lateral sinus; if the base of the skull, the orbit, the mouth, the jaw, or the nose is affected, it will be the cavernous sinus.

When thrombosis occurs with meningitis the lesions are much the same as in the cachectic form, with the exception that there are sometimes slight changes in the walls of the sinuses. If the patient has suffered from a local septic process, there may be puriform softening of the clot, and general pyæmia, with the development of secondary abscesses in the brain, in the lungs, and in other organs. With such cases there may be associated a general or localised meningitis.

*Symptoms.*—The symptoms of septic thrombosis are more decided than those of the cachectic form. When occurring in the course of meningitis, it usually adds no new symptoms to those of the original disease. In the pyæmic form the symptoms are more characteristic, particularly when associated with otitis. There are recurring chills with very high and widely fluctuating temperature. There is headache, and often localised tenderness of the scalp; the other symptoms which are present are usually the same as those of meningitis. If metastasis occurs, there may be evidences of abscesses in the brain or in other organs, and sometimes there are signs of suppuration in the jugular vein. A polymorphonuclear leucocytosis is usually present, and blood cultures in most cases show the presence of pyogenic organisms.

The local symptoms of the thrombosis differ somewhat according to the sinus affected: if its seat is the superior longitudinal sinus, there may be cyanosis of the face, dilatation of the temporal and frontal veins, and sometimes epistaxis; if the lateral sinus is involved, the process may extend to the jugular vein, which may be felt in the neck as a hard cord, and there may be dilatation of the veins of the mastoid region, and even localised œdema; when the cavernous sinus is affected, there may be protrusion of the eyeball of the affected side, œdema of the lid, and with the ophthalmoscope the retinal veins appear enlarged and tortuous, sometimes being the seat of thrombosis. The process may affect either one or both sides. The course of septic thrombosis is rather irregular, varying from a few days to three weeks. In fatal cases death takes place from meningitis, cerebral abscess, or pyæmia. The prognosis is very grave, unless the disease is so situated that it is accessible to surgical operation.

*Treatment.*—The only successful treatment is surgical. Operation is easiest in thrombosis of the lateral sinus, being much more difficult if involving the superior longitudinal sinus. So many cases are now on record of successful operation upon septic thrombosis of the lateral sinus, that it should always be urged when the diagnosis is clear.

#### CEREBRAL ABSCESS.

Cerebral abscess is quite rare in children, decidedly more so than is cerebral tumour. In Gowers' collection of 223 cases, only twenty-four

were under ten years of age. In infants, abscess is one of the least frequent diseases of the brain, and up to five years it is exceedingly rare.

**Etiology.**—By far the most frequent cause in children is otitis. This is the origin of the great majority of the cases. Abscess rarely complicates acute otitis, but is seen with the chronic form. Exactly how otitis causes cerebral abscess it is not always easy to determine. Usually there is caries of the petrous bone, but there may be none. The infection may extend through the small veins traversing this bone, or along the lateral sinuses to the cerebellum. Abscess is often attributed to the retention of pus in the ear, but it may occur when the discharge is free.

Traumatism is the second important etiological factor. Abscess may be associated with fracture of the skull, or follow simple concussion. The abscess is generally in the neighbourhood of the injury, but occasionally is produced by *contre coup*. In one instance, reported by Wagner, thrush was believed to be the cause of cerebral abscess, the same fungus that existed in the mouth being found in the brain, which in this case was studded with small abscesses. Abscesses may be the result of infectious emboli, associated with general pyæmia, though this is rare in early life; and finally it may occur without any assignable cause.

**Lesions.**—The most frequent seat of the abscess is, first, the temporo-sphenoidal lobe; secondly, the cerebellum; thirdly, the frontal lobes. Other locations are very rare. Abscesses are usually single. In size they vary from that of a small cherry to an orange. One case was observed by Meyer, in which an abscess occupied one entire hemisphere. The contents are usually thick greenish-yellow pus, which may be very fetid. When abscesses have lasted for some time they are usually surrounded by a dense pyogenic membrane, and may become encysted. The pathological process may be slow, and often is apparently stationary for a long period. Abscesses may rupture into the ventricles, less frequently upon the surface of the brain, causing meningitis, or the pus may even escape externally through the auditory meatus.

**Symptoms.**—These are general and local. The general symptoms are much the more important for diagnosis, and often are the only ones present. The local symptoms are those of a tumour. The clinical history of a case of abscess of the brain may be divided into three stages: First, the period of onset, or early acute inflammatory symptoms, fever, etc., which attend the formation of pus. Secondly, the latent period, or period of remission, in which very few symptoms are present. In many acute cases this stage is wanting altogether; in the chronic cases it may last for months, or even years. Thirdly, the final period, with recurrence of active cerebral symptoms, followed by death in a few days.

The onset may be accompanied by symptoms so slight as almost to escape notice. In most cases, however, headache and fever are present. The headache is usually severe, and often localised upon the affected

side; in cerebellar abscess it may be occipital. The fever is moderate in intensity, and continuous. In addition there may be vertigo, vomiting, general convulsions, and cessation of the aural discharge, if one has been present. The duration of this stage is variable; it may be only a few days, or several weeks. It is shorter in traumatic cases, and in those which are due to pyæmia.

The latent stage, or period of remission of symptoms may be quite short—only a few days' duration—and it is often absent. During this period the temperature may fall quite to the normal, and the headache disappear, or be only occasional and slight. However, if any focal symptoms have been present they remain unchanged.

The symptoms of the terminal stage are due to a rapid extension of the inflammatory process, with œdema and softening about the abscess, sometimes to rupture into the ventricle, and sometimes to meningitis. The fever now returns, and may be high. There is headache, often very intense and continuous; there may be delirium and convulsions, and the gradual development of coma. In addition there may be vomiting, paralysis, opisthotonus, retracted abdomen, and the other symptoms of meningitis. Occasionally all the earlier symptoms may be latent, and the terminal symptoms may be the only ones present. In infants, the fontanel is usually large and bulging; convulsions are rather more frequent than in older children.

The local symptoms of abscess are rather indefinite, owing to its usual situation. Abscesses of considerable size may exist in the temporo-sphenoidal lobe, in the central part of the frontal lobe, or in the cerebellum, without any definite local symptoms. If the abscess is near the motor area, there are the usual symptoms of disease in this location: spasm, or paralysis of the face, arm, or leg. A cortical or sub-cortical abscess is likely to cause convulsions. Cerebellar abscess may give rise to occipital headache, frequent vomiting, and when the abscess is large enough to press upon the middle lobe, there may be inco-ordination of the muscles of the extremities. Optic neuritis may be present, but other symptoms relating to the cranial nerves are rare. Localised tenderness over the scalp, when persistent, is a symptom of importance, and may serve to locate the abscess, if it is superficial.

**Diagnosis.**—Of the general symptoms, the most important for diagnosis are fever, headache, delirium, and terminal coma. These become particularly significant when following otitis or traumatism. The differential diagnosis of abscess is to be made principally from tumour and meningitis, and from these conditions more by the history and general course of the disease than by any special symptoms. The diagnosis of abscess from tumour is considered in connection with the latter disease. It is more difficult to distinguish between meningitis and abscess, since the two processes are often associated. With meningitis convul-

sions are more common, but they are rarely localised; rigidity and the inflammatory symptoms are more intense; the course is usually more rapid and more regular, being rarely interrupted, as is the course of abscess. Leucocytosis is more constant and usually more marked in meningitis. Lumbar puncture gives negative results in uncomplicated abscess while it gives positive definite information in meningitis. From the cerebral symptoms occurring with otitis it is extremely difficult to distinguish abscess, for optic neuritis may be present in the former as well as in the latter condition. The more intense and prolonged the cerebral symptoms and the more marked the neuritis, the greater are the probabilities of abscess.

**Prognosis.**—The prognosis in cerebral abscess is always grave, unless accessible to surgical operation. The progress may be slow, or rapid, but it is inevitably from bad to worse, and sooner or later the disease, if not interfered with, proves fatal.

**Treatment.**—The medical treatment of abscess in its active stage is that of any acute intracranial inflammation—ice to the head, absolute quiet, free catharsis, and full doses of the bromides or morphine, if pain is intense. The absolutely hopeless condition of these cases when left to themselves, and the recent brilliant results from surgical operations, should lead the physician to urge operation in every case.

#### CEREBRAL TUMOUR.

Very little has been added to our knowledge of cerebral tumour in children since the exhaustive monograph of Starr; to this I am indebted for many of the facts in this chapter.

**Varieties and Location.**—Tumour of the brain is not very infrequent, and may be seen even in infancy. From this time up to puberty there is no period of special susceptibility. In 269 of the cases in Starr's collection, in which the nature of the tumour was stated, the following were the varieties:

|                    |            |
|--------------------|------------|
| Tubercle .....     | 152 cases. |
| Glioma .....       | 37 "       |
| Sarcoma .....      | 34 "       |
| Glio-sarcoma ..... | 5 "        |
| Cyst .....         | 30 "       |
| Carcinoma .....    | 10 "       |
| Gumma .....        | 1 case.    |
| — — — — —          | 269 cases. |

Tuberculous tumours are more often multiple than are other varieties. Their most frequent seat is the cerebellum; next to this the pons and crura cerebri. They are rarely cortical or central. Glioma is most often found in the cerebellum or in the pons, and next in the cortex; but it is

rarely central. Sarcoma is most frequently in the cerebellum; next to this, in the order of frequency, in the pons, the basal ganglia, and the cortex. Cystic tumours are either central or cerebellar. Taking the cases as a whole, the most frequent seat of tumour in children is, first the cerebellum, second the pons, third the centrum ovale.

Tuberculous tumours are occasionally seen in infancy, but they occur most frequently between the ages of five and twelve years. They are usually secondary to tuberculosis elsewhere, especially in the lungs and in the bronchial lymph nodes. They most frequently start from the membranes, rarely being centrally situated, and extend inward, infiltrating the superficial portion of the cerebellum or cerebrum. There is almost invariably localised meningitis at the site of the tumour; there may be adhesions between the dura and pia mater, and the disease may extend to the cranial bones. In size, these tumours vary from a small pea to a child's fist. They may be softened and broken down at the centre, or cheesy throughout. They are the result of a localised tuberculous inflammation, which does not differ essentially from that seen in other parts of the body.

Glioma is not infrequent in infancy. It is probably connected in every case with the ependyma of the ventricle. It repeats the structure of the neuroglia, being composed of connective tissue and branching cells.

Sarcoma may be of the spindle-celled or the mixed variety. It grows much more rapidly than glioma. The two varieties are not infrequently combined in the same tumour—glio-sarcoma.

Cystic tumours are sometimes sarcomatous in origin, the wall of the cyst containing sarcoma cells, and they may also be parasitic, from the growth of the echinococcus. They may be found in any part of the brain.

The other varieties of sarcoma, gummata, and vascular tumours, are exceedingly rare until after puberty.

As the tumour grows, secondary lesions are produced in most of the cases. These are the result of pressure upon arteries, causing localised anæmia, or even cerebral softening; or upon veins, producing congestion and œdema. When affecting the middle lobe of the cerebellum, pressure upon the venæ Galeni may lead to effusion into the ventricles. Localised meningitis over tumours superficially situated is the rule, and this may be the cause of some of the symptoms. Rarely, cerebral hæmorrhage may be associated.

**Etiology.**—The causes of cerebral tumours are for the most part unknown. In a few instances there is a history of definite traumatism. Sarcoma or carcinoma may be secondary, and tuberculous tumours are probably always so.

**Symptoms.**—These may be divided into two groups: first, the general symptoms which are common to tumours of all varieties, and are in-



dependent of location; secondly, the local symptoms depending upon the situation of the growth.

*General Symptoms.*—One of the most frequent is headache. Though it varies much in its severity, character, and position, it is rarely absent. It is apt to be severe, and may continue for a long period, or it may be intermittent. The location of the pain has no definite relation to the situation of the tumour. It may be accompanied by sensations of tightness, compression, or tension in the head. It may be associated with localised tenderness of the scalp; when this is constant it is a valuable symptom for diagnosis, as it often occurs with tumours superficially located.

General convulsions are frequent in the early stage, but separated by quite long intervals; they become more frequent and more severe as the disease progresses. All degrees of severity are seen, from slight twitchings and temporary loss of consciousness, to typical epileptiform seizures. They are most common when the growth is rapid and when complicating meningitis is present. Attacks of vomiting or of localised spasm may for a considerable time precede general convulsions; and in a single attack there may be first localised and then general convulsions.

Mental symptoms are generally present in great variety and complexity. There may be only fretfulness and irritability, or a marked change in disposition. These symptoms are so frequent from other causes in children that they excite no apprehension, unless to them are added dulness, apathy, and somnolence. Later in the disease there may be attacks of hypochondriasis, or of melancholia; there may be periods of wild, almost maniacal excitement; and, finally, the mental impairment may approach a condition of imbecility.

Optic neuritis and optic-nerve atrophy are very frequent, occurring, according to Starr, in eighty per cent of the cases. This is only recognised by the ophthalmoscope, as there may be no disturbance of vision. The optic neuritis is generally double, appears earlier, and is more constant in basal tumours than in those at the convexity, or those centrally located.

Vomiting is very frequent, but diagnostic only when it occurs suddenly without assignable cause, and without nausea or other symptoms of indigestion. It is especially significant when frequently repeated, and of more importance in older children than in infants.

Vertigo is often associated with vomiting. At first it is occasional and seen upon changing position, but later it may be quite constant, especially with tumours in the posterior fossa.

Disturbances of sleep are frequent. There is usually insomnia, but sleep may be broken by hallucinations, accompanied by attacks of screaming; rarely is there persistent drowsiness until toward the end of the disease.

*Local Symptoms.*—These depend upon the situation of the tumour, but not at all upon its character. Local symptoms may be wanting entirely, and they may vary much in different cases even with tumours in the same situation. They are modified by the size and by the rapidity of growth, and by the existence of localised meningitis.

In tumours of the cortex, the meninges are likely to be involved, especially with tuberculous and gliomatous growths. The pathological process may extend from within outward or from without inward. The most frequent general symptoms in such cases are headache, circumscribed tenderness of the scalp, convulsions, and mental symptoms. Optic neuritis, vomiting, and vertigo are not so common. Tumours situated in the frontal lobe, as a rule, present few symptoms and may be entirely latent. Irritation of the frontal lobe may extend to the motor area and cause convulsions either local or general; but not often is there paralysis. Tumours of the left side (of the right side in left-handed persons) in the third frontal convolution may cause motor aphasia.

Tumours in the motor convolutions along the fissure of Rolando produce the most definite and uniform local symptoms. When situated at the upper portion the leg is affected, at the middle portion, the arm, and at the lower, the face. Irritative symptoms, such as rigidity or clonic spasm, commonly precede for some time the paralysis which results from pressure or destruction. These attacks of localised convulsions may begin in the face, arm, or leg; but they usually extend more or less rapidly until all three are involved. There is no loss of consciousness, but there may follow a slight transient paralysis. Such attacks are known as "Jacksonian epilepsy," and form one of the most diagnostic symptoms of cerebral tumour. Localised spasm may be associated with anæsthesia or other disturbances of sensation. The paralysis generally first affects one extremity—the arm or leg, according to the location of the tumour—and afterward it may involve the entire side, including the face.

If the tumour is centrally located, or at the base, hemiplegia may be an early symptom from pressure on the motor tract. With cortical paralysis there may be associated ataxia and anæsthesia.

Tumours of the parietal lobe may give no local symptoms. At times there are disturbances of muscular sense, tactile sensibility, or sensations of pain and temperature. If the inferior parietal lobule of the left side is affected, there may be word-blindness, or inability to understand written language.

Tumours of the occipital lobe produce, as the only constant local symptom, hemianopsia. This is usually bilateral, affecting the same side of both eyes, being on the side opposite to that of the lesion, i. e., a tumour on the right side causes blindness in the left half of both eyes, so that the patient sees nothing to the left of a line directly in front

of him. Instead of hemianopsia, there may be only irritation and various disturbances of sight.

Tumours of the temporo-sphenoidal lobe may be latent, or, if on the left side, may cause word-deafness, i. e., inability to understand the significance of spoken language.

Tumours in the island of Reil when situated upon the left side (right side in left-handed persons) may cause motor aphasia or disturbances of speech. If they are large they may produce symptoms by pressure upon the motor tract—hemiplegia or monoplegia.

Tumours of the basal ganglia cause marked general symptoms, but none of a definitely local character. The important symptoms relate to the various tracts or bundles of fibres which pass from the cortex through the internal capsule. These include the motor and the various sensory tracts, the olfactory, auditory, visual, and speech tracts. Any of these may be pressed upon, and the nature of the symptoms will depend upon the size of the tumour and the extent of the pressure. If only the anterior part of the capsule is affected there may be no symptoms; if the middle fibres, hemiplegia and disturbances of articulation; if the posterior fibres, hemianæsthesia. All these may be associated, and any of them may be complete or partial. Tumours in this situation are apt to implicate the cranial nerves. Optic neuritis is quite constant, and appears early. Localised or general convulsions are rare.

The peculiar symptoms pointing to tumours of the crura cerebri are nystagmus, strabismus, and loss of pupillary reflex, sometimes with general muscular inco-ordination, and a staggering gait. There is usually third-nerve paralysis on the side of the tumour, and on the side opposite to the hemiplegia with which it is often associated. This variety of crossed paralysis is quite diagnostic. The symptoms of third-nerve paralysis are external strabismus, dilatation of the pupil, and ptosis. In these cases optic neuritis appears early. There may be a complicating hydrocephalus. While hemiplegia is commonly present with large tumours, it may be absent with small ones, or may appear later than paralysis of the third nerve.

Tumours of the pons are quite common. The diagnostic symptoms consist in crossed paralysis, the cranial-nerve symptoms being on the side of the tumour, and the general motor and sensory symptoms on the opposite side. When the seat is the upper half of the pons, the third and fifth nerves are apt to be implicated, giving rise to ptosis, dilatation of the pupils, external strabismus, trophic disturbances such as ulceration of the cornea, and neuralgic pain in the face. Tumours in the lower half of the pons involve the sixth, seventh, and eighth nerves, causing internal strabismus, contracted pupils, facial paralysis, sometimes deafness, and auditory vertigo. Other symptoms associated with tumours of the pons are headache, vomiting and optic neuritis; convulsions being rare.

Tumours of the medulla are recognised by the involvement of the glossopharyngeal, pneumogastric, spinal accessory, and hypoglossal nerves. There are difficulty of deglutition, irregular respiration, irregular pulse, and vaso-motor disturbances, such as flushing of the face and perspiration. There may be projectile vomiting, polyuria or glycosuria, opisthotonus, difficulty in articulation or in sucking, and in protrusion of the tongue. When large, these tumours may produce symptoms of pressure upon the motor or sensory tracts—paralysis, or partial anæsthesia, with rigidity and exaggerated reflexes.

Tumours of the cerebellum are especially important, this being the most frequent location in childhood. When only one hemisphere is affected there may be no local symptoms. Tumours involving the middle lobe, or those large enough to produce pressure upon the middle lobe, give rise to vertigo and cerebellar ataxia. Vertigo is especially frequent; it may occur with headache. Cerebellar ataxia is different from the ataxia due to a spinal-cord lesion, and strikingly resembles that of intoxication. It may increase until the patient is unable to walk, although there is no loss of muscular power. Vomiting is a frequent symptom, as are also optic neuritis, and headache which is usually occipital. When there is secondary hydrocephalus, as is not uncommon, mental symptoms are present, and there may be enlargement of the head. Opisthotonus is occasionally seen, but general convulsions are rare.

**Diagnosis.**—The size of the tumour is to be determined mainly by the general symptoms, special attention being given to the order of their development. A diagnosis as to the nature of the tumour is really not of much importance; but some information upon this point may be gained from the consideration of its etiology, the rapidity of its growth, and the age of the patient. Cerebral tumour may be confounded with abscess, tuberculous meningitis, chronic basilar meningitis, and chronic hydrocephalus. The symptoms distinguishing tumour from abscess are the following: Tumour may occur at any age; without definite etiology, excepting when tuberculous; the progress is steady, but generally slow, new symptoms being continually added; headache is more constant and more severe; optic neuritis more frequent; cranial nerves more often involved; mental disturbances more marked; focal symptoms are often definite; fever and leucocytosis are absent; duration, six months to two years. As compared with the above, abscess is not so frequent, being especially rare in infancy; there is a definite history of traumatism or ear disease; progress more irregular; symptoms often intermittent; headache less severe; mental symptoms less marked; optic neuritis and involvement of the cranial nerves less frequent; focal symptoms usually indefinite; localised tenderness over the scalp more constant; fever and leucocytosis present except in the latent period; the most frequent complication is acute meningitis.

Cases of tuberculous meningitis which may be confounded with tumour are those of slow course sometimes seen in older children. The difficulty in diagnosis is increased by the frequent association of tuberculous tumours with tuberculous meningitis. The main points of difference are that in tumour the symptoms are more localised and the course generally much slower. Almost every individual symptom, however, may be present in the two conditions.

Chronic basilar meningitis may produce symptoms almost identical with those of tumour in the posterior fossa. It is, however, confined to infancy; hydrocephalus and opisthotonus are much more marked than are usually seen with tumour.

Chronic hydrocephalus may resemble tumour; this occurs so frequently as a lesion secondary to tumour that the question often arises whether there is only hydrocephalus, or there is in addition a tumour. Primary hydrocephalus is usually congenital, and commonly attains to a greater degree than is seen in secondary hydrocephalus.

**Prognosis.**—The prognosis in cerebral tumour, while bad, is not hopeless. Cases are occasionally seen which exhibit all the characteristic symptoms of tumour, even including optic neuritis, which recover perfectly. These are probably syphilitic, although often no such history can be obtained. In other cases, most frequently of a tuberculous nature, an arrest of the growth occurs and the patient recovers with some function of the brain impaired; usually there is loss of vision or some paralysis. In most cases, however, the progress is steadily downward until death.

**Treatment.**—If there is any reason to suspect syphilis, the iodide of potassium should be given in large doses and continued for a long period; the effect of this drug even in tumours not syphilitic is sometimes beneficial. Starr refers to a case in which symptoms of six months' duration, including optic neuritis, entirely disappeared under the use of mercury and the iodide. The tumour was supposed to be gumma, but an autopsy obtained six months later showed it to be a sarcomatous cyst. For a discussion upon the surgical aspect of the treatment of brain tumours, the reader is referred to Starr's work on Brain Surgery.

#### HYDROCEPHALUS.

Hydrocephalus, or "water on the brain," consists in an accumulation of serum in the cranial cavity. This may be between the dura mater and the pia (external hydrocephalus) or in the ventricles of the brain (internal hydrocephalus). The former is secondary and is quite rare, while the latter is not uncommon. Hydrocephalus may be acute or chronic.

**Acute hydrocephalus** is secondary to basilar meningitis, which is usu-

ally of tuberculous origin. The terms tuberculous meningitis and acute hydrocephalus are sometimes used synonymously. A moderate distention of the ventricles is frequent in all varieties of acute meningitis. The amount of fluid in acute hydrocephalus is not great, there being rarely more than three or four ounces present.

**Chronic external hydrocephalus** except in its mild form is extremely rare, and is nearly always a secondary lesion. It may follow meningeal hemorrhage, pachymeningitis, or any lesion causing cerebral atrophy. It is seen in its most marked form associated with congenital malformations of the brain, particularly imperfect development of the hemispheres. (See Fig. 123.) On incising the dura mater a few ounces, or



FIG. 123.—BRAIN IN EXTERNAL HYDROCEPHALUS, SHOWING IMPERFECT DEVELOPMENT OF THE HEMISPHERES. Patient three and a half months old; head measured  $20\frac{1}{4}$  inches; increase in size, 2 inches in the six weeks before death; symptoms were typical of ordinary internal hydrocephalus. In the picture the small size of the cerebrum is best judged by comparison with the cerebellum, which is normal. The hemispheres were rudimentary; the basal ganglia were normal; the cranial cavity contained about one pint of fluid.

sometimes even a pint, of serum may escape. The convolutions are somewhat flattened, and may be greatly atrophied. Other lesions are found either in the brain or in the dura mater. External hydrocephalus may cause enlargement of the head and separation of the sutures, and in fact most of the symptoms of the internal variety; but usually it is not severe enough to give rise to any decided symptoms.

## CHRONIC INTERNAL HYDROCEPHALUS.

This is the important variety, and when no qualifying term is mentioned this is the form of hydrocephalus which is always understood.

**Etiology.**—This occurs both as a primary and a secondary condition. When secondary it is usually associated with tumours of the base of the brain or with chronic basilar meningitis, either simple or tuberculous. It is in these cases a mechanical condition caused by pressure which obliterates the openings from the lateral ventricles into the fourth ventricle, or the foramen of Magendie.

The causes of primary hydrocephalus are as yet very little understood. In a large proportion of the cases the disease is congenital, generally beginning in the latter months of intra-uterine life. Some of these cases are clearly syphilitic. Rickets and hydrocephalus are occasionally associated, but so infrequently as to make a definite etiological connection between them very doubtful. The rachitic head has been so often mistaken for hydrocephalus that an erroneous notion has arisen as to the frequent association of these two diseases. Heredity is a factor of some importance; numerous instances are on record where two children in the same family have been affected. Hydrocephalus not infrequently develops after successful operations upon spina bifida or encephalocele.

**Lesions.**—The difference between the primary and secondary cases is chiefly one of degree. The amount of fluid in secondary cases is rarely more than three or four ounces. In primary cases it is usually from half a pint to one pint, but it may be very great. In one of my own cases there was removed from the head of a child, who died at four months, five pints of fluid. Larger quantities than this have been reported, but not so far as I am aware at so early an age. In composition this resembles the cerebro-spinal fluid. An examination in one of my cases showed it to be a clear, translucent fluid, slightly alkaline in reaction, specific gravity 1.005, containing sodium and potassium chlorides, alkaline phosphates, and a trace of albumin. In some specimens sugar is found. In cases of inflammatory origin the amount of albumin is generally larger, and the fluid may be slightly turbid. The effusion may become purulent from accidental infection resulting from operation, from rupture, or, as in one of my cases, from infection through the sac of a spina bifida with which it was complicated, the process extending to the brain through the central canal of the cord.

The changes in the brain result from the gradual accumulation of fluid in the ventricles. The septum lucidum is usually broken down, and all the avenues of communication between the ventricular cavities are greatly enlarged. The continuous distention results in a gradual thinning of the brain substance which forms the ventricular walls; often these are found only one-fourth of an inch in thickness, or even less

than this, the cortex being a mere shell (Fig. 124). In one of my autopsies the ependyma of the ventricle and the pia mater were in places actually in contact, all of the brain tissue having been absorbed; the brain resembled a large double cyst. In a case of Peterson's, with the exception of a small portion of one temporo-sphenoidal lobe, all of both hemispheres had disappeared, the cerebellum and basal ganglia alone being intact. The brain is always anæmic, and the gray and white substance may be indistinguishable. The changes are largely mechanical, the microscope showing, in my case just referred to, only granular matter and round nuclei evidently from broken-down nerve cells. In less

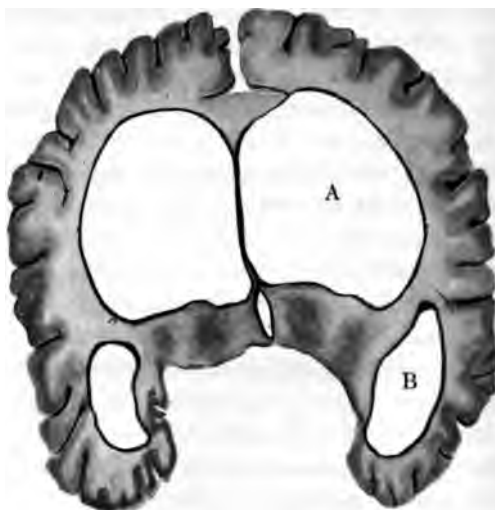


FIG. 124.—VERTICAL TRANSVERSE SECTION OF A BRAIN IN CONGENITAL HYDROCEPHALUS. From a child who died at the age of three weeks. A, distended lateral ventricle; B, its descending horn.

severe cases the changes may be slight. It is, however, always surprising to see the amount of compression which the cortex will tolerate without interference with its functions, provided the pressure comes gradually. The ependyma may be normal, but it is usually somewhat thickened and pale, sometimes granular, and may be infiltrated with new cells. When infection takes place an acute ependymitis may be set up. Chronic inflammation of the ependyma is thought to be the essential lesion in many of the primary cases, whether of simple or syphilitic origin.

The bones of the skull are markedly affected; the sutures at the vault are widely separated, and sometimes even those at the base. After the removal of the fluid the head collapses, giving an appearance which has been well likened to a "bag of bones." It should not be forgotten, however, that hydrocephalus may coexist with premature ossification, in which case the head may be small. In the cases which recover, the wide gaps in the skull may be closed by the development of Wormian bones; but ossification is often not complete until the fifth or sixth year.

The most frequent lesion associated with congenital hydrocephalus is spina bifida, in which case there may also be a patency of the central canal of the spinal cord; more rarely meningocele or encephalocele are met with. Sometimes there are deformities in other parts of the body, such as club-foot or hare-lip.



**Symptoms.**—Hydrocephalus may exist with a small head. In this condition there is usually premature ossification of the cranial bones. Four such cases have come under my notice, one child having lived to be fourteen months old. These children are usually idiotic, and die at an early age, often from convulsions. In such cases other malformations of the brain are frequently associated.

Hydrocephalus, with the exceptions mentioned, is recognised by the increased size of the head. In order to estimate the amount of enlargement, it must be remembered that at birth the circumference of the normal head is about 14 inches, and at one year from 18 to 19 inches. The degree of enlargement in hydrocephalus may be very great. In one of my cases, the head at four months measured  $24\frac{1}{2}$  inches. In another at ten and a half months,  $26\frac{3}{4}$  inches. Steiner has reported a remark-



FIG. 125.—CHRONIC HYDROCEPHALUS OF AVERAGE SEVERITY.  
Head of pyramidal shape; showing characteristic expression of the eyes.

able case in which the head at eight months measured  $32\frac{3}{4}$  inches. When the enlargement of the head is not great the diagnosis is not so easy. Hydrocephalic enlargement is commonly symmetrical and in all directions. The head is sometimes globular in outline and sometimes pyramidal (Fig. 125). The forehead is exceedingly high and projecting, and there is a prominence of the frontal eminences seen in no other

form of enlargement. The sutures may be separated from half an inch to two or three inches; the fontanel is very large, tense, and bulging; the veins of the scalp are enlarged and prominent. In marked cases fluctuation may be readily obtained, and the head may even be distinctly translucent.

In the acquired form all these symptoms are less marked, and if ossification of the skull has taken place it is often impossible to discover any increase in size. The rate of growth of the head varies much in different cases, and it is the surest measure of the progress of the case. The increase in circumference is usually from one to three inches a month.

The primary cases are for the most part of congenital origin, and the child may die *in utero*. At other times the process may have advanced so far before birth that puncture of the head is necessary before delivery is possible. In perhaps the majority of cases no symptoms are observed at birth, or the head is only slightly larger than normal. Usually nothing is noticed until the child is two or three months old, when it is discovered that the head is increasing in size at an abnormal rate. If the progress is rapid, other symptoms are soon evident: the infant can not hold up his head; he is lethargic, and all his perceptions are dulled, sight and hearing included; there may be a general flaccid condition of all the muscles of the extremities due to a slight general paresis, but more often there is rigidity, which is usually most marked in the legs, but sometimes in the arms; the hands are often clenched, with the thumbs adducted; the reflexes are exaggerated; the pupils are generally contracted and equal, though they may be dilated; nystagmus and convergent strabismus are often present. Convulsions may occur from time to time, or may be deferred until near the close of the disease. As the head enlarges the body usually wastes, and the disproportion between the two may seem greater than it really is.

Such congenital cases rarely see the end of the first year, and are often fatal during the first six months. The causes of death are marasmus, convulsions, and intercurrent disease, rarely rupture of the head.

In the cases which develop more slowly, the symptoms are quite different. The head may not attain at eighteen months the size reached in the other cases at the third or fourth month. The surprising thing about many of these cases is that the distinctly cerebral symptoms are so few. When the pressure develops gradually, the brain seems able to tolerate an almost indefinite amount of it. The more readily the bones of the skull yield to pressure the fewer are the nervous symptoms; hence, other things being equal, they are less marked when the disease begins before the sutures are firmly ossified than in the later cases. A comparatively small amount of effusion may cause very marked symptoms in a child two or three years old, while a much larger amount, in

an infant of a year, may produce much less disturbance. It is for this reason that secondary hydrocephalus causes such striking symptoms, although the accumulation of fluid is small.

Whether the progress of these cases is slow or rapid, the development of the children is greatly retarded. Many are not able to support the head until two or three years old; frequently they do not walk until five or six years old. The special senses are generally not noticeably affected, but intelligence in most cases is interfered with—in some only slightly, in others very markedly, while some are idiotic. Contractions of the extremities are occasionally seen, but usually more of the hands than the legs. Sensation is not often affected. The course is a very chronic one. From time to time there are exacerbations of the symptoms, and even intercurrent meningitis may be excited.

**Prognosis.**—Most of the congenital cases are fatal before the end of the first year. It is very rare that a hydrocephalic child reaches the age of seven years. The process may, however, go on up to a certain age and then cease spontaneously, and the child may go through life with a head very much larger than normal and usually with a mental condition somewhat impaired. Retrogression of the symptoms is, however, never to be looked for.

**Diagnosis.**—The most important symptom is the enlargement of the head, and this can only be arrived at by careful measurement and comparison with the normal size. The rapidity of growth is quite as important for diagnosis as the fact of enlargement. If the head grows as much as an inch a month there can be little doubt. The enlargement most frequently confounded with hydrocephalus is that which occurs in rickets. In the latter disease it is almost invariably irregular; there are prominences over the two frontal eminences and over the parietal bones, often with furrows between them; the size of the head is chiefly due to thickening of the bones of the skull; the marked prominence of the forehead is not seen, and the increase in the bi-parietal diameter is not present; furthermore, there are other signs of rickets.

**Treatment.**—If there is any suspicion of syphilis, mercurial inunctions should be employed, and potassium iodide given internally in full doses. Of all the operative measures that have been proposed for this condition, and their name is legion, the only one at the present time which seems to hold out any reasonable prospect of permanent improvement is auto-drainage. This consists in establishing a communication between one of the lateral ventricles and the sub-arachnoid space. By this means the fluid is conducted to a place from which it can be absorbed. A considerable number of cases have now been treated in this way. The dangers of the operation are considerable, nearly half the patients having died as the direct result of it. Of those who have survived, a number have shown improvement and a few very striking improve-

ment, but no complete cures have been reported. Operation is not to be recommended in early cases with rapidly increasing enlargement. The best results have been obtained in old cases which have reached a nearly stationary condition.

### INFANTILE CEREBRAL PARALYSIS.

(*Spastic Diplegia, Paraplegia, or Hemiplegia.*)

Under the term cerebral paralysis are included several groups of cases with causes quite dissimilar, but having certain definite clinical features in common. While the symptomatology is quite clear, there are many questions relating to the pathology that are not yet fully settled, although much has been added to our knowledge within the last few years. Paralysis depending upon cerebral tumour, abscess, or hydrocephalus is not included in this chapter.

The cases of cerebral paralysis may be divided into three groups, according as the paralysis depends upon conditions existing prior to birth, upon those connected with birth, or upon those of subsequent development.

**I. Paralysis of Intra-Uterine Origin.**—This is the least frequent condition. In such cases there is some congenital defect in the brain, due sometimes to arrest of development, at others to such intra-uterine lesions as hæmorrhage or thrombosis. There may be porencephalus, or cysts extending deeply into the substance of the brain, sometimes communicating with the ventricles. The origin of this condition is for the most part unknown. In rare cases the paralysis is due to cortical agenesis,<sup>1</sup> a condition in which the brain may seem normal to the naked eye, but the microscope shows a complete arrest in the development of the cells of the cortex, usually affecting both hemispheres. In still other cases there are found gross defects in development in the motor centres of the cortex. Such a lesion is shown in Fig. 137. Cases in which there is conclusive evidence of intra-uterine hæmorrhage are very rare.

**Symptoms.**—In most of the paralysees due to intra-uterine lesions, loss of power is only one of the symptoms, and usually not the most prominent. It is rare that there is not some mental impairment, and usually idiocy is present. The type of paralysis is nearly always diplegic or paraplegic. When this is due to arrested cortical development, a general flaccidity of the muscles may be seen instead of the rigidity so characteristic of the other forms of cerebral paralysis.

**II. Birth-Paralysis.**—Cerebral birth-paralysis is due in nearly all cases to meningeal hæmorrhage. The primary lesions and the early symptoms have already been described in connection with the Diseases of the Newly Born. The secondary lesions present considerable variety.

<sup>1</sup> For fuller description, see Sachs' Nervous Diseases of Children.

There may be found (1) meningo-encephalitis, (2) atrophy and sclerosis of the cortex, (3) cysts upon the surface, (4) secondary degenerations in the spinal cord.

1. *Meningo-encephalitis*.—This lesion is often quite diffuse. There is thickening of the pia mater, and it is usually adherent to the brain substance. The cortex is involved to a variable degree, depending some-



FIG. 126.—EXTENSIVE ATROPHY AND SCLEROSIS OF THE RIGHT HEMISPHERE. From an infant seven and a half months old; probably the result of a meningeal hæmorrhage at birth (lateral view). *History*.—Twelve hours after birth was seized with general convulsions, which continued for three days. No other symptoms noticed till one month before death, when weakness of the left arm was observed. Never held head erect. Was plump and well nourished; died from erysipelas. *Autopsy*.—Pia not adherent; a large cyst occupied the region of the occipital and posterior part of the parietal lobes, showing in its floor discolouration and pigmentation, evidently from an old hæmorrhage. Right optic nerve, tract, and crus much smaller than the left.

what upon the time which elapses between the initial lesion and the autopsy. The following were the microscopical changes found by Sachs<sup>1</sup> in the brain of a child in my wards at the Babies' Hospital, who died at the age of one year of measles: The lesions were found everywhere in the cortex. The pia was universally adherent, and showed general cellular infiltration; its blood-vessels showed marked cell proliferation, and the veins in the sub-pial space were dilated and filled with blood. In the pia dipping in between the convolutions similar changes were present. In the cortex few, if any, normal pyramidal cells were found,

<sup>1</sup> The clinical features of this case are quite as interesting as the pathological findings. The child was a first-born, delivered after a dry labour of forty-eight hours. It was asphyxiated, and from the first days of its life it had attacks of convulsions, usually repeated many times a day. During one of these convulsions the photograph from which Fig. 127 was made, was taken by Dr. Peterson. The child had the symptoms of typical spastic paraplegia—the arms being, however, slightly involved—retarded mental development, and convergent strabismus.

but in the outer layers were an enormous number of small glia cells. Many of the blood-vessels showed a cell-proliferation of their walls. There was also degeneration in the pyramidal tracts of the lateral columns of the cord.

2. *Atrophy and Sclerosis*.—These changes vary much in extent and degree. There may be only a circumscribed area in which the convolutions are small, firmer than usual, and covered with an adherent pia, or there may be an atrophy so extensive as to involve a large part of one hemisphere (Fig. 126), or sometimes of both hemispheres. Usually the lesion is somewhat diffuse over the convexity of both sides, and much more frequently of the anterior than of the posterior half of the brain. Where a depression of the brain exists the space is filled with cerebrospinal fluid, and in many cases there is a deformity of the skull.

3. *Cysts upon the surface* may occur alone or in connection with the lesions just mentioned. These are usually small, about the size of a walnut, but they may cover a large part of a hemisphere. Such large cysts are sometimes classed as cases of external hydrocephalus.

4. *Secondary degenerations* of the internal capsule and the lateral columns of the cord are found in most of the cases associated with extensive atrophy and sclerosis, and in many of those in which only meningo-encephalitis is present.

*Symptoms*.—The type of paralysis will, of course, depend upon the extent and position of the original lesion. A diffuse lesion is followed

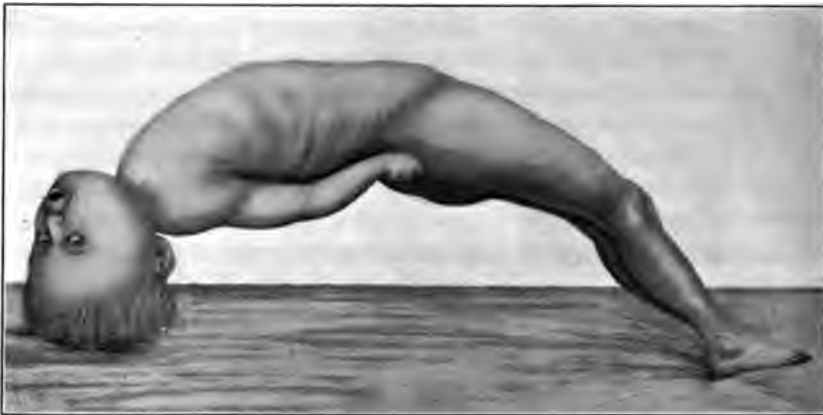


FIG. 127.—CONVULSIONS IN SPASTIC PARAPLEGIA.  
From a photograph by Dr. Frederick Peterson during an attack.

by diplegia; one not quite so extensive by paraplegia; one affecting one side only, by hemiplegia, or even monoplegia, though this is very rare. The relative frequency of the different forms will vary according to the age at which the patients come under observation. According to my

own observations, which have been chiefly upon infants, the cases of diplegia and paraplegia have outnumbered those of hemiplegia more than four to one. My belief is that the great majority of the congenital cases, or those due to hemorrhage occurring at birth, are diplegias or paraplegias, and that very many of them succumb during the first two years; however, the cases of hemiplegia, because of the less serious lesion, live much longer. Diplegia and paraplegia will therefore be considered as the characteristic types of cerebral birth-palsy, as the cases of hemiplegia do not differ from those due to later causes—i.e., the acquired form.



Fig. 1. A young child with cerebral birth-palsy, showing the characteristic posture of the arms and legs.

The child in the photograph is standing with its arms held out to the sides, a common posture in children with cerebral birth-palsy.

In the most severe cases that survive the symptoms of the early days of life there remains some rigidity of the extremities, chiefly of the legs, which is constant or intermittent, slight or well marked. There is often spasm of the muscles of the neck and trunk, giving rise to convulsions. In many cases there are frequent attacks of convulsions of the face. The general physical development of the child is often retarded, with a child being smaller and weaker than normal. One of some acute cases of birth-palsy, never having been able to stand or even stagger, died at the age of five years. In other cases the general intelligence is not affected, and life may be prolonged considerably, but usually with some degree of mental impairment. The degree of impairment may be slight or of all degrees; it may be noticed at birth or noticed until the child is some years old, or the child may be normal until three or four years of age, and then gradually become insane until they are some years old, and do not walk again. In some cases the child is normal until some years old, and then gradually becomes insane. The children in the photograph are of the type of cerebral birth-palsy, and are of the type of cerebral birth-palsy, and are of the type of cerebral birth-palsy.

In the milder cases the early symptoms may be overlooked, and nothing excite suspicion until the infant is six or eight months old. There is then discovered unmistakable muscular weakness; the child does not sit up, or even hold up the head when the trunk is supported. Often there is observed before this time a tendency to stiffen the body and to throw the head backward, owing to spasm of the cervical or spinal muscles. The muscular weakness is often mistaken for rickets, or regarded simply as backwardness. A closer examination usually discloses the presence of some rigidity of the extremities, particularly of the legs, and exaggeration of the knee-jerks. As the child grows older other symptoms of imperfect development become more and more evident.

There are changes in the shape of the skull, this being usually smaller than normal in all its diameters, or there may be asymmetry. There is an arrest of development in the paralysed limbs. These are both smaller and shorter than normal. In many cases abnormal movements are seen, which may be of an irregular choreic type, or they may be athetoid. Epilepsy develops in from thirty-three to fifty per cent of all these patients.

**III. Acute Acquired Paralysis.**—This is usually of the hemiplegic type, although diplegia and paraplegia may in rare instances be met with. This group includes cases developing at any time after birth, but the great majority of those seen in childhood begin before the fifth year.

*Etiology.*—The etiology is often obscure. The paralysis sometimes follows traumatism. It is occasionally seen in the course of scarlet fever, measles, diphtheria, variola, or pneumonia. Much more frequently than with any of these diseases it occurs during pertussis, being usually the outcome of a severe paroxysm. The frequency with which these cases are ushered in with convulsions has led many to assign this as the cause of the paralysis. It is probable that the convulsions are more often the result than the cause of the lesion. In some of the acute inflammatory cases the cause is possibly the same as in acute poliomyelitis.

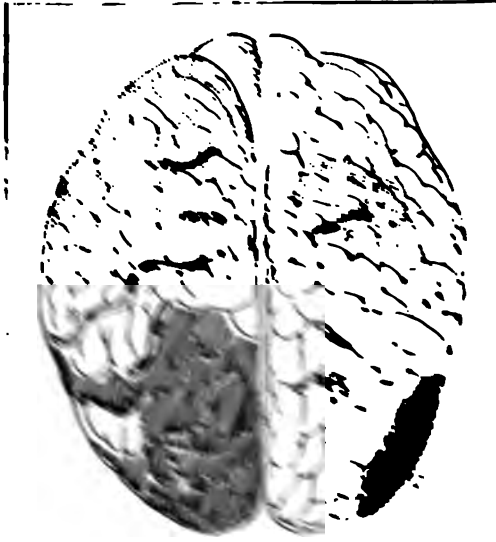
*Lesions.*—The lesions of acute cerebral palsy may be grouped under three heads: (1) those of the blood-vessels; (2) those of the membranes; (3) those of the brain substance.

1. Lesions of the Blood-vessels.—There may be hæmorrhage, embolism, or thrombosis. Hæmorrhage is by far the most important. It is usually meningeal, rarely cerebral. It occurs more frequently at the convexity than at the base, and is often diffuse. Meningeal hæmorrhage may result from pachymeningitis. It may be due to traumatism, when it is also from the dura mater; or from the acute hyperæmia accompanying paroxysms of pertussis, when it may be from the dura or the pia; or it may be secondary to thrombosis of the superior longitudinal sinus.



The association of hemorrhage with sinus-thrombosis is not very infrequent. It was found in one of my autopsies upon a patient who died of pneumonia. Cerebral hemorrhage is extremely rare, but it occurs

even in infants; I once saw it in one only two months old.



Embolism is rarely found unless associated with acute rheumatic endocarditis, and then usually in children who are over seven years old. As a rule, the usual seat of the embolism is a branch of the cerebral artery. The embolus has been met with in a small branch of a vessel, but is more common in the main trunk.

The embolism of the cerebral arteries is usually associated with acute rheumatic endocarditis, and then usually in children who are over seven years old. As a rule, the usual seat of the embolism is a branch of the cerebral artery. The embolus has been met with in a small branch of a vessel, but is more common in the main trunk.

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Acute paralysis sometimes occurs for which no explanation can be found at autopsy. An infant with pneumonia was admitted to the Babies' Hospital, who had developed, a few days before, typical right hemiplegia. It came on suddenly, with convulsions, and involved the face, arm, and leg. The arm and leg appeared to be completely paralysed, but in the face the paralysis was incomplete. The paralysis had begun to improve somewhat at the time of the child's death, which occurred a little over a week after its onset. At the autopsy no gross lesion could be discovered. A careful microscopical examination was made, and nothing abnormal was found except a slight increase of small spheroidal cells about some of the meningeal and cortical vessels of the motor area. The frontal and occipital lobes were normal.

*Symptoms.*—While diplegia and paraplegia are occasionally seen, the great majority of cases of acquired cerebral palsy are of the hemiplegic variety. When diplegia and paraplegia occur, it is usually in early infancy, and their symptoms and course differ in no wise from the birth palsies. We may therefore regard hemiplegia as the chief manifestation of acquired cerebral palsy.

The onset of the paralysis is almost invariably sudden, with convulsions, which are usually repeated, and in severe cases followed by loss of consciousness. In the secondary cases these are generally the only symptoms. In one of my cases the patient went to bed apparently well, and awoke in the morning with hemiplegia. Such an onset, however, is very exceptional.

When the paralysis is due to acute poliomyelitis, the onset is usually with high fever, vomiting, often convulsions, followed by delirium or stupor. These general symptoms continue for a variable time, usually two or three days, before paralysis is seen. The temperature in most cases is from 101° to 103° F., and the fever sometimes follows, sometimes precedes, the convulsions. The loss of consciousness may last for several days, and the paralysis is frequently not discovered until consciousness is regained. If there is a very extensive lesion there may be diplegia, deep coma, and death, but this is very infrequent. Usually the lesion is more limited, and the symptoms are those of typical hemiplegia. The face sometimes escapes, and if involved it generally soon recovers. The paralysis of the arm and leg is at first complete, but may improve rapidly in the course of a few weeks. Disturbances of sensation may be present, but are usually of a transient character. After a variable period, from one to several weeks, the patient begins to use the paralysed extremities, first the leg, afterward the arm, as in adult hemiplegia. The convulsions may be repeated for the first day or two, but prolonged or continuous convulsions are rare. They may be general or unilateral. With lesions of the left side of the brain, speech may be affected, and

not infrequently in young children when the lesion is upon the right side. The reflexes are increased upon the affected side, and a slight ankle-clonus may be present.

After a few weeks the child may be able to walk, dragging the affected leg. The recovery in the leg is sometimes complete, but in most cases a slight halt in the gait remains. The arm usually recovers more slowly than the leg, and contractures are likely to develop after a variable



FIG. 130. — DEFORMITY OF LEFT HAND THE RESULT OF CONTRACTURES FOLLOWING AN ATTACK OF HEMIPLEGIA FOUR YEARS BEFORE. Child seven years old.

time, generally two or three years. In Fig. 130 is shown a frequent deformity of the upper extremity. Contractures of the leg lead to various forms of talipes, generally equinus, from shortening of the tendo-Achillis. Sometimes the arm or the leg recovers so perfectly that the case may be regarded as one of monoplegia. In old cases the paralysed limbs are atrophied; there is more or less rigidity, and the spastic condition may be quite marked. I have seen this limited to a single group of muscles in the leg. Aphasia is common in right hemiplegias, and it is not very rare in those of the left side, because infants appear to use both sides of the brain with nearly equal facility.

The mental condition of these children is often normal, in striking contrast with the cases of congenital diplegia. The earlier the paralysis occurs the more likely are mental symptoms to be present, since we have here not only the direct effect of the lesion, but an arrested development of some part of the brain. Epilepsy is not an uncommon sequel; it may be of the Jacksonian type, or there may be attacks of general convulsions. In other cases

there are post-hemiplegic movements of a choreic or athetoid character, or irregular inco-ordinate movements.

**Prognosis of Infantile Cerebral Paralysis.**—In diplegia and paraplegia the outlook is always unfavourable. A very large number of these cases which are due either to intra-uterine or birth lesions never reach the third year, but die in infancy from marasmus or acute intercurrent disease. Those who survive usually show serious mental defects, and

many are practically helpless on account of the extreme spastic condition of the muscles of the extremities.

In hemiplegia the prognosis is much more favourable. In most of these cases the paralysis is of the acute acquired variety, and the later the period of onset, the less likely is the brain to be seriously damaged. In some of these patients complete recovery takes place; in others the residual paralysis is so slight as to be easily overlooked except on careful examination, the occurrence of epilepsy being perhaps the first thing which leads one to suspect that a previous paralysis has existed. The great majority of children who have suffered from infantile cerebral palsy have some degree of permanent paralysis and usually some deformities from contractures, the extent of both varying, of course, with the severity of the primary lesion. In all cases seen in young infants it is exceedingly difficult to give a prognosis in regard to future mental development. As a rule, the impairment is directly proportionate to the extent of the paralysis and its intensity.

**Diagnosis.**—The diagnosis between the congenital and acquired forms of cerebral palsy is of no great practical importance, and it may be impossible; for the symptoms in congenital cases are often not sufficiently marked to attract attention until children are old enough to sit alone or to walk.

It may be quite difficult to distinguish cerebral paralysis from infantile spinal paralysis. The history of an acute onset, the atrophied limbs, the deformities, and the absence of sensory disturbances, may be found in both conditions. Spinal paralysis is, as a rule, monoplegic, and often affects but a single group of muscles. Cerebral paralysis is either diplegic or hemiplegic in character, and even though only a leg or an arm may seem to be affected, a critical examination will usually reveal the fact that the other limb of the same side has also suffered. The presence of rigidity and exaggerated reflexes is quite as important evidence of this as loss of power. The electrical reactions, however, are conclusive; the reaction of degeneration is absent in cerebral paralysis, while it is present in spinal paralysis.

Simple as the differentiation may seem in most cases, the mistake is frequently made of confounding cerebral diplegia, particularly of the flaccid type, with rickets. Cases of acute acquired paralysis at the onset may be mistaken for acute meningitis, but early loss of consciousness, the early development of the paralysis, its permanent character, and the shorter duration of the acute symptoms, usually distinguish these cases from those of meningitis. The only definite means of differential diagnosis is by lumbar puncture; this gives negative results in cerebral paralysis and positive results in meningitis.

**Treatment.**—The course and the result of cerebral paralysis depend upon the extent of the injury to the brain, its nature, and the age at

which it is inflicted—all these being conditions which are beyond the power of the physician to modify or control. The treatment of cerebral palsy is therefore extremely unsatisfactory. For the congenital cases practically nothing can be done, except for the deformities and complications. The acquired cases during the acute onset are to be managed like all other cases of acute cerebral congestion or inflammation—absolute rest, ice to the head, and bromides. Electricity is not to be used in early cases, and little or nothing is to be expected from it in the late ones. Much can be accomplished in an educational way for the mental derangements resulting from cerebral palsy. An important part of the treatment relates to the deformities. Many of these may be prevented by the early use of orthopædic apparatus. Serious deformities in old cases may be greatly benefited by tenotomy or myotomy, followed by the use of suitable apparatus. Division of the posterior nerve roots has been performed for the relief of extreme spasticity with, in some cases, very striking benefit. Epilepsy is to be treated as when it depends on other causes.

#### MENTAL DEFECTS.

##### DEFICIENCY, IDIOCY, IMBECILITY.

All grades of mental defects are seen in children. While the terms above used characterise the chief clinical types, it should be remembered that these shade into each other by almost imperceptible degrees. They may be the result either of arrested development or of disease or injury of the brain.

The backward child does not belong in this group, although often placed here by parents or teachers. Such children may present many mental peculiarities, but differ from the normal standard chiefly in the slowness with which the mental functions are developed, the most noticeable of these being speech. It is backward children and those who present the milder grades of mental defect that are of the greatest clinical interest and importance, for in them the mental condition often depends upon some physical cause which time and proper treatment may remove. Common causes are defective sight or hearing, severe early rickets, prolonged malnutrition, etc.

Following somewhat the classification of Ireland, the mental defects of children may be divided into the following groups:

1. Those depending upon such congenital conditions as porencephalus, arrested development of the brain as a whole, or of some portion, particularly the frontal lobes. An excellent illustration of this class of cases is seen in Fig. 137. Another variety is known as "Agnesia corticalis," described elsewhere.
  2. Those associated with external or internal hydrocephalus.
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3. Those associated with microcephalus, either with or without premature ossification of the cranial bones (Figs. 134–136).

4. The paralytic cases, including the varieties which occur in the different forms of cerebral paralysis, the greater part of which are due to meningeal hæmorrhage at birth, and which are clinically associated with

VARIOUS TYPES OF MENTAL DEFECTS.



FIG. 131.



FIG. 132.



FIG. 133.

FIGS. 131–133.—Mongolian type.

FIG. 131.—Six months old; died at twenty-two months; could not hold up the head, or understand anything.

FIG. 132.—Boy six and a half years old; did not walk or talk till four years old; now quite intelligent, but not normal.

FIG. 133.—Girl four years old; mental development like that of a normal child of two and a half years; walks very awkwardly.



FIG. 134.



FIG. 135.



FIG. 136.

FIG. 134.—Boy twelve years old; microcephalic; walked at about four years; can read and write; development like that of a normal child of eight years.

FIG. 135.—Microcephalic, seven years old; understands most of what is said; can not talk intelligibly.

FIG. 136.—Girl of eight years; imbecile; can not walk without help.

Note that the expression in 132, 133, and 134 is not due to adenoids; 132 and 134 have had them removed.

spastic diplegia or paraplegia; a smaller number are associated with acquired cerebral paralysis, most frequently following meningeal hæmorrhage.

5. Those of inflammatory origin. They follow cerebro-spinal meningitis and acute poliomyelitis.

6. Those associated with epilepsy, in which the condition is a result

of changes in the brain produced by the repetition of the epileptic seizures.

7. Mongolian Idiocy.—This is a form characterised by a peculiar Chinese type of skull and face, with marked backwardness in physical and mental development (Figs. 131–133). The head is somewhat flattened from before backward; the nose rather broad and flat; but the



FIG. 137.—ARRESTED DEVELOPMENT OF THE FRONTAL LOBES OF THE BRAIN, PARTICULARLY OF THE RIGHT SIDE. From an idiotic child twelve months old.<sup>1</sup>

most striking thing is the narrow palpebral fissures which have a downward inclination toward the nose. These patients almost always have the mouth open; and the facial expression like that due to large adenoids may lead to the suspicion that this is the only condition present. The mouth breathing is, however, due rather to the peculiar conformation of

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<sup>1</sup> A microscopical examination by Dr. Martha Wollstein showed the cortex in the affected region to be only one-third the normal thickness; the cortical layers were ill-defined; there was a striking absence of the characteristic nerve cells, both the large and small pyramidal cells being few in number. There was no growth of connective tissue. The white substance was normal, as were also the dura and pia.

the base of the skull, and the anterior projection of the bodies of the upper cervical vertebræ. The Mongolian type is seen in all degrees of severity. In early infancy these children may present no striking peculiarities except in facial expression, and a general backwardness in physical development. Dentition is delayed; they may not sit alone until the age of eighteen months or two years, and frequently do not walk or talk intelligently until they are four or five years old. In the milder forms they are often regarded simply as very backward children. In the more severe forms the mental defect may be great. Their resistance is feeble, and many die in early childhood. Little is known of the etiology of this condition. Cases occur in all classes of society, and when other children in the family are quite normal.

8. Amaurotic Family Idiocy.—This name, proposed by Sachs, indicates the prominent features of the malady, which is not a very rare one. Nothing is known of its etiology except that nearly all the recorded cases have been in the Jewish race. Two, and sometimes three or four children in succession have been affected in the same family. The first symptoms are usually noticed between the sixth and tenth month, up to which time the infant has generally appeared normal. At first it is only noticed that the child is making no progress in his development. He does not gain in ability to sit up or use his muscles in other ways. He lies quietly, does not respond as he once did, and takes less interest in his surroundings. After a few weeks it is clear that the child, instead of advancing, is actually retrograding, both physically and mentally. His muscles become so weak that he can no longer sit up or even hold up his head. Closer observation shows that vision is becoming less and less distinct. The child no longer recognises the faces of friends or objects shown him. Finally, he becomes dull, apathetic, and quite indifferent to his surroundings, and it is evident that he can not see at all. In the early stages the muscles are usually weak and flaccid; later there is rigidity, with increased knee-jerks and often marked spasticity. There may be general convulsions. The characteristic features of the disease are revealed by the ophthalmoscope. There is a milky-blue or white area, with a bright, cherry-red centre, occupying the place of the macula lutea, and with this there is also atrophy of the optic disc. The ocular changes are symmetrical. The disease is progressive, accompanied by marked wasting, and usually fatal within a year from the time when the first symptoms are seen; but occasionally the blind, helpless child may live for two or even six years. The essential lesion consists in degenerative changes of the ganglion cells of the central nervous system. The changes are most marked in the cerebral cortex, but are widespread, and hardly a normal ganglion cell may be found. The outlook is absolutely bad, all cases terminating fatally.

9. Both sporadic cretinism and chondro-dystrophy have many symp-



toms suggesting mental defects, but they do not strictly belong in this category. They are considered separately later.

In addition to the etiological factors belonging to the different conditions above described, the influence of heredity is to be considered; there may be hereditary nervous diseases, alcoholism, syphilis, or some other vice of constitution. Intermarriage among blood relations is one of the causes most frequently assigned; but after an exhaustive study of the question, Huth reaches the conclusion that this view is not supported by the facts.

**Diagnosis.**—Certain types of mental defect may easily be recognised after the age of three or four years, especially the more marked forms when they are due to the graver cerebral lesions—hydrocephalus, microcephalus, various cerebral palsies, amaurotic idiocy, etc. In the milder forms and in infancy, however, this is not so easy a matter; it is often impossible without a considerable period of observation to distinguish a backward or peculiar child from one who has some serious mental defect.

To appreciate the abnormal, one must be familiar with the mental and physical development of healthy children. A normal infant of average muscular development can usually support the head steadily before five months old, often at three months; he can usually sit erect at eight or nine months, and stand with assistance at twelve or thirteen months. Toys are held and usually handled with facility at five or six months. The recognition of the nurse or mother comes at about the same time. Usually the first distinct words are pronounced about the end of the first year, and at two years most children put words together in short sentences. Variations of a few months from the averages above mentioned can not be considered abnormal.

To determine whether an abnormal mental state is simply the result of poor general nutrition, or is dependent upon actual disease or imperfect development of the brain, is frequently a matter of the greatest difficulty. The backward infant is usually distinguished chiefly by the things which he does not do; while with those who are deficient not only are the proper signs of development wanting, but many new and peculiar symptoms may be observed. The backward child may not sit alone until he is twelve or fifteen months old, and may not walk until he is two and a half years old, but the cerebral development is in most cases proportionate to the physical condition. Speech may be so delayed that the first words do not come until two years, and short sentences not until three years old, and yet in understanding what is said to and done for him, the child may seem bright and his development steady and progressive, although slow.

All children whose development is delayed should be examined for local signs of cerebral disease; the symptoms mentioned under the vari-

ous heads of early hydrocephalus, meningeal hæmorrhage, and cretinism should be sought. Sight and hearing should be tested, and the eyes, if possible, examined with an ophthalmoscope; the co-ordination of the hands should be tested in various ways; the reflexes examined, and general rigidity or slight paralysis noted, also the muscular power in the trunk, neck, and extremities. Many children who are mentally deficient do not show any disturbances of nutrition during the first year. The growth of the body in height and weight may be quite normal; although this is rarely true of the muscular power. Some of them show marked signs of backwardness in physical development, and in nearly all there are some other striking symptoms. Among the most frequently noticed are: drooling, an open mouth, a protruding tongue, a fixed, aimless stare, the production of some inarticulate sounds, which are usually peculiar to the child and may be repeated many times a day. Occasionally there are sharp screams without any evident cause, also irregular, aimless movements of the hands. Objects are not properly held, and if grasped, they are soon dropped by an infant of twelve or fourteen months as by a normal one of three or four months. The child does not recognise his bottle or his nurse. Nystagmus is often present; and there may be ill-defined attacks of a convulsive nature, or typical convulsions. The infant is not attracted by bright colours or toys, and, in short, seems dull and unresponsive to every mental impression.

An accurate diagnosis usually carries with it the data for a definite prognosis. Few misfortunes which can befall a family are worse than to have a mentally defective child, and the physician's opinion is sought early and eagerly as to the probable outlook for all children who are suspected of being in any way abnormal. The possibilities of error in the early years are great, and much needless suffering is often caused to parents by an erroneous opinion. It is the experience of all who see many of these children, that some who were regarded at the age of three or four years as seriously defective, have in the end turned out to be entirely normal. One should therefore always put the best possible interpretation upon the facts. The amount of improvement which takes place in many of these cases is most surprising. The above statement applies, of course, chiefly to children in whom there are no evidences of gross cerebral lesions. The deviations from what is normal are many and wide, and careful observation for a long period is necessary before a child is pronounced idiotic or even feeble-minded.

Most cases of idiocy exhibit to a greater or less degree the stigmata of degeneration. In an examination of 517 idiots by Howe, there was found blindness in 21; deafness in 12; some defect of the nose or mouth, such as hare-lip, high palatal arch, or cleft palate, in 23 cases; and some deformity of the hands or feet in 54 cases; while in 96 there was paralysis of one or more limbs.

**Treatment.**—The problem is essentially an educational one, and for such education special teachers and often special schools are indispensable. With such advantages it is surprising to see what can be accomplished with many children who have a severe grade of mental defect. To furnish a proper means for educating these children is a duty of the State, and up to the present time very inadequate provision has been made for them. Except in the mild forms, defective children are better trained and educated in institutions than in the home, and parents should be urged to place them in institutions whenever practicable as soon as they have passed the age or development of infancy.

#### CHONDRO-DYSTROPHY.

*(Achondroplasia—Congenital or Fœtal Rickets.)*

This rather rare condition is the cause of some of the most marked examples of dwarfism known. It was recognised as an abnormality by the early Egyptians and has figured in art in various ways since that date.



FIG. 138.—SKULL IN CHONDRO-DYSTROPHY, SHOWING FRONTAL PROMINENCE AND PROGNATHISM. Girl six years old.

Paintings show that many of the old court jesters were of this type. Because of their striking appearance, these dwarfs have always excited much curiosity and interest.

The causes of chondro-dystrophy are unknown; only in rare cases has any hereditary connection been traced. The pathological process begins in foetal life and consists in a disturbance of the normal ossifica-

tion of primary cartilage. It affects endochondral ossification only, never intra-membranous ossification. The flat bones and the vertebræ therefore escape, while the bones of the extremities suffer most. The disease does not affect bones which are cartilaginous or almost entirely so through the greater part of intra-uterine life. One of the most striking changes in the skull is the synostosis or early ossification of the tribasilar bone; this is formed of two parts of the sphenoid and the sphenoidal process of the occipital bone. Normally this ossification does not take place until adult life; in children with chondro-dystrophy it often begins *in utero*. This prevents a normal expansion at the base of the skull, and the brain, as it grows, is thus crowded upward and forward, causing the great prominence of the forehead (Fig. 138). The upper jaw appears very prominent on account of the depression at the root of the nose.

In the long bones there is a marked interference with the normal row-formation of the proliferating cartilage cells, which may be seen in all degrees. In some cases a periosteal lamella pushes its way between the epiphysis and the diaphysis, still further restricting the growth of the long bones. As bone formation beneath the periosteum goes on normally, the bones in this condition are thick as well as short.



FIG. 139.—NORMALLY DEVELOPED LONG BONES OF A FÆTUS COMPARED WITH THOSE OF CHONDRO-DYSTROPHY. (Spillmann.)



FIG. 140.—CHONDRO-DYSTROPHY—INFANTILE FIGURE. (Marie.)

**Symptoms.**—The majority of children suffering from this condition are either born dead or die shortly after birth. Those who survive are delicate during infancy, but afterward may become strong and healthy. The most striking thing about their appearance is the very short legs and arms as compared with the length of the body. At birth the arms in many cases do not reach to the waist line, and the length of the body may be less than the circumference of the head. The epiphyses appear

somewhat enlarged, the abdomen is prominent, the skin of the extremities is in deep folds, the soft parts seeming to be much too abundant for the shortened bones (Fig. 140). In infancy these children are often quite fat. The facial expression is characteristic. There is usually a deep depression and flattening at the base of the nose, with a very marked prominence of the forehead. The head may not only seem large, but by measurement may be one or even two inches above the normal average. An erroneous diagnosis of hydrocephalus is often made in the early stage. Dentition is slightly later than normal, but not more so than is seen in moderate rickets. Marked relaxation of the ligaments and rather feeble muscular power often delay walking until the third or fourth year. If the head is large, the fontanel may not close till the fourth or fifth year. The appearance of the fingers is quite characteristic, causing the so-called "trident hand." The fingers are very short and of nearly equal length, and an angular separation is seen at the second joint (Fig. 141).



FIG. 141.—CHARACTERISTIC HAND OF CHONDRO-DYSTROPHY. (Marie.)



A B  
FIG. 142.—A. NORMALLY DEVELOPED BOY, AGE EIGHT YEARS. B. TYPICAL CHONDRO-DYSTROPHY, AGE EIGHTEEN YEARS. (Marie.)

Although not normal in their mental development, these children are far from being feeble-minded. They are often several years behind the normal in speech and in most intellectual efforts. The average patient is able to read and do many ordinary things, but throughout life always remains somewhat peculiar, and on critical examination is found to be subnormal in his mental growth. These dwarfs are good-natured, often amusing, easily controlled, and frequently live to a great age. With advancing years the figure assumes a very peculiar and characteristic appearance. The prominent hips, with the marked lordosis, shortened

extremities, and late bowing of the legs, present a striking picture (Fig. 142). The maximum height attained is often not more than three and a half or four feet. Although while young of feeble muscular power, later in life they often become very muscular. When adult life is reached the sexual powers are normal; if the women become pregnant, Cæsarian section is almost always required on account of deformity of the pelvis.

In infancy, chondro-dystrophy is often confounded with rickets, hydrocephalus, and cretinism; but its features are so characteristic that the mistake can hardly be made if the child is carefully examined. No known treatment has any influence upon the condition. The use of the thyroid extract is entirely without effect.

### SPORADIC CRETINISM.

(*Cretinoid Idiocy; Myxædematous Idiocy.*)

Since the early description of this disease by Fagge, in 1871 and 1874, numerous cases have been published in England, on the continent of Europe, and in America, showing that sporadic cretinism is not confined to any country. While the condition is relatively a rare one, since it has been generally recognised it is found to be much more common than was formerly supposed.

**Etiology.**—It is now well established that this condition depends upon the absence of the internal secretion of the thyroid gland. In a series of sixteen autopsies collected by Fletcher Beach, the thyroid gland was absent in fourteen and the seat of bronchocele in two. The symptoms closely resemble the myxædema of adults which follows the removal of the thyroid. Regarding the causes which destroy the thyroid gland or abolish its functions little is as yet known. In most cases it is probably a congenital condition. In some instances it has followed acute disease. In a certain number of cases sporadic cretinism is associated with goitre. As a rule, only one case occurs in a family, the other members of which present nothing abnormal in mental or physical development.

**Symptoms.**—The symptoms of cretinism in most cases make their appearance during the first year, but are sometimes so slight as not to be noticed until children are two or three years old, and exceptionally not until the seventh or eighth year. The general appearance of the cretin is striking, and so characteristic that when once seen the disease can hardly fail to be recognised (Figs. 143, 144, and 146). The body is greatly dwarfed, and children of fifteen years are often only two and a half or three feet in height. All the extremities, the fingers and the toes, are short and thick. The subcutaneous tissue seems very thick and boggy, but does not pit upon pressure like ordinary œdema. The facies

is extremely characteristic: The head seems large for the body; the fontanel is open until the eighth or tenth year, and it may not be closed even in adults; the forehead is low and the base of the nose is broad, so that the eyes are wide apart; the lips are thick, the mouth half open, and the tongue usually protrudes slightly; the cheeks are baggy, the hair coarse, straight, and generally light-coloured. The teeth appear very late—in one of my cases none were present at two years—and are apt to decay early.

Fatty tumours are quite constant in older children, although they are often wanting in infantile cases. They are seen in the supra-clavicular region, just behind the sterno-mastoid muscle, sometimes in the axilla, or between the scapulæ, and sometimes in other parts of the body. In distribution they are apt to be symmetrical, and are usually about half the size of a hen's egg. The neck is short and thick. In rare cases there may be a slight depression corresponding to the location of the thyroid

gland. The chest is not deformed. The abdomen is large, pendulous, and resembles that of rickets. An umbilical hernia is almost always present. The skin is dry, perspiration scanty, and eczema is common. The voice is hoarse and rough. Patients often do not walk until they are five or six years old, and then they waddle in a clumsy way. All the movements of the body are slow and lethargic, and everything indicates mental and physical torpor. The rectal temperature is usually subnormal. I had once an opportunity to observe an attack of acute broncho-pneumonia in one of these cretins two years old. The symptoms and physical signs were typical, but during the greater part of the disease the rectal temperature fluctuated between 95° and 98.5° F. Only once was a temperature above 99° F. recorded. On account of their low temperature and torpid condition these patients are very sensitive to cold. The mental condition is always impaired, and they are often idiotic. Speech is



FIG. 143.—A TYPICAL CRETIN; TWO AND A HALF YEARS OLD. A patient in the Babies' Hospital.

acquired late, and in some cases not at all. Cretins are dull, placid, and good-natured, rarely troublesome or excitable; and when fifteen or eighteen years old they appear like children of two or three years. There is

an absence of development of the sexual organs, and almost invariably they suffer from chronic constipation.

**Diagnosis.**—The diagnosis is usually easy, although the early cases are sometimes miscalled rickets. The low temperature, the facial expression, the torpor, and the fatty tumours are enough to differentiate the two diseases.



FIG. 144.—DR. J. P. WENT'S CASE OF CRETINISM, SEVENTEEN MONTHS OLD, BEFORE TREATMENT.



FIG. 145.—AFTER SIX MONTHS' TREATMENT WITH THYROID EXTRACT.

**Prognosis and Treatment.**—There is little tendency to spontaneous improvement. Many of these patients die in childhood, but a few live to adult life. Until within the last few years they were considered hopeless. The thyroid extract is a specific remedy for this disease. In many cases the improvement is truly remarkable (Figs. 144–147). After a few months' treatment the entire appearance of the child is changed. The idiotic expression of the face is lost; the thickening of the skin and subcutaneous tissues disappears; there is a marked increase in height and in the circumference of the head; muscular power is rapidly developed, so that many soon become able to walk; and progress is seen in dentition, and in some older girls in the establishment of menstruation. Intellectual progress is much slower than physical changes; how-



ever, nearly all the children become brighter and more intelligent and learn to speak.

The ultimate results vary with the grade of the affection and the time when treatment is begun. I have under observation several cretins who have been treated from eight to twelve years. Although many of



FIG. 146. — DR. J. W. COYNER'S CASE, TWENTY-THREE MONTHS OLD, BEFORE TREATMENT.



FIG. 147. — AFTER ELEVEN MONTHS' TREATMENT WITH THYROID EXTRACT.

these children seem quite intelligent and are able to attend school, they are without exception below other children of their ages in mental and physical development. Complete recovery I have not seen; but there seems to be no reason why it might not occur if the thyroid were begun in early infancy and faithfully continued. If the thyroid is omitted, relapses occur in a few months, even in cases well advanced toward recovery.

Most of the thyroid extracts on the market are prepared from the glands of the sheep. There is little doubt that the fresh glands are more active than the extracts prepared from them; but they are difficult to obtain. A reliable extract should be given if results are to be expected. The thyroid extract of Burroughs and Wellcome I have found to be more satisfactory than many of those on the market. Of this half a grain

may be given once or twice a day at first; after the child becomes somewhat accustomed to it the daily dose may be gradually increased to five or six grains. Some disturbances are often seen at the beginning of the treatment—perspiration, marked irritability, and sometimes a rise in temperature—but these soon pass off. For old cases at least five grains daily should be given for an indefinite period.

## INSANITY.

Insanity is so special a subject that all that will be attempted here will be to mention the most frequent varieties seen in early life, with the important etiological factors which operate at this period. For a full discussion of the subject the reader is referred to works upon insanity.

Insanity is distinguished from idiocy in that it affects a mind previously sound; however, the two conditions may be associated. Undoubted cases of mental disease have been observed before the seventh year, but they are extremely rare. From this time up to puberty, however, nearly all the varieties seen in adult life occasionally occur, but they are very infrequent even at this period. The form which insanity in childhood most frequently assumes is mania.

**Etiology.**—Insanity is sometimes seen as a sequel of one of the infectious diseases, more often typhoid fever than any other, although it may follow measles, scarlet fever, diphtheria, or variola. Another cause is masturbation, although its effect is much more frequently seen after puberty than before. Hereditary syphilis is sometimes the cause of dementia, which comes on about the fourth or fifth year, or even later. Alcoholism, epilepsy, insanity, or other nervous diseases in the parents are important causes. Prolonged or continuous mental strain, the result of overwork in school, is a cause of considerable importance, especially in girls about the time of puberty. As exciting causes may also be mentioned various reflex conditions, such as intestinal worms, phimosis, delay in the establishment of menstruation, and abnormal conditions of the nose and throat; these, however, can not have much influence except where the predisposition is a strong one. Insanity may be associated with or may follow hysteria, chorea, or epilepsy. It has sometimes followed injury to the brain, acute meningitis, and occasionally other forms of brain disease.

**Symptoms.**—Certain forms of insanity are practically never seen in children, such as paranoia, acute dementia, paretic dementia, periodic or circular insanity, and cataleptic insanity.

Mania is one of the most frequent forms, and is the most common variety of post-febrile insanity. Its symptoms may be quite intense, but are usually of short duration, lasting but a few days or weeks. In rare cases it may continue for months, and it may even be permanent.

Melancholia is not uncommon. It is seen as a result of prolonged mental strain in school, it may be due to fear of punishment, and sometimes may follow masturbation. It is usually associated with some very marked disturbance of the general health. It shows itself, as in the adult, by fits of depression, self-mutilation, and even by suicidal tendencies.

Epileptic insanity may follow epilepsy in children who were previously mentally sound, in whom it may take the form of true epileptic dementia, or there may be attacks of mania which occur in the place of an epileptic seizure or follow such a seizure. Transitory attacks of fury or frenzy coming on without apparent cause should always suggest the possibility of epilepsy.

Other forms which insanity assumes in early life are: transitory psychoses, such as delirium, night-terrors, attacks of sobbing or weeping, sometimes from fright; moral insanity, as shown by perversion of the moral sense and by various vicious tendencies; morbid impulses, which may be homicidal or sexual, or a disposition to thieving, lying, pyromania, etc.; morbid fears, of which there may be an almost endless variety. These are sometimes associated with a low state of physical health; this, however, is usually not the case.

**Prognosis.**—On the whole, insanity in childhood has a better prognosis than in the adult. In most of the cases of mania, melancholia, the various transitory psychoses, or the choreic and hysterical forms, recovery occurs with proper treatment. The outlook for the other varieties is much worse, especially in those in which there is a strong hereditary tendency to mental disease.

The treatment is to be conducted along the same general lines as in adults.

#### THE STIGMATA OF DEGENERATION.

These marks are of much importance in relation to the different forms of nervous disease in children, especially epilepsy, idiocy, and insanity. They are of great value in determining existing nervous disease, or as showing latent neuropathic tendencies.

The physician should be familiar with these various signs in order that he may connect them with each other and refer them to their proper source, and at the same time, by appreciating their significance, be able to advise parents with regard to the care, education, mode of life, and occupation of children, in whom to a greater or less degree these signs may be present. These stigmata are not of equal importance as marks of degeneration. Some of them, such as facial asymmetry and most of the deformities of the palate, are always to be so regarded; the speech defects are often so, while many of the others may or may not be, according to their association. The stigmata are divided into anatomical,

physiological, and psychical. The following is the classification given by Peterson:

**Anatomical Stigmata.**—Cranial anomalies: Facial asymmetry; deformities of the palate; anomalies of the teeth, tongue, lips, or nose.

Anomalies of the eye: Flecks on the iris; strabismus; chromatic asymmetry of the iris; narrow palpebral fissure; albinism; congenital cataract; pigmentary retinitis.

Anomalies of the ear.

Anomalies of the limbs: Polydactyly; syndactyly; ectrodactyly; symelus; phocomelus; excessive length of the arms.

Anomalies of the trunk: Herniæ; malformation of the breasts and thorax; dwarfishness; giantism; infantilism; feminism; masculinism; spina bifida.

Anomalies of the genital organs.

Anomalies of the skin: Polysarcia; hypertrichosis; absence of hair; premature grayness.

**Physiological Stigmata.**—Anomalies of motor function: Walking late; tics; tremors; nystagmus; epilepsy.

Anomalies of sensory function: Deaf-mutism; neuralgia; migraine; hyperæsthesia; anæsthesia; blindness; myopia; hypermetropia; astigmatism; Daltonism; hemeralopia; concentric limitation of the visual field.

Anomalies of speech: Mutism; defective speech; stuttering; stammering.

Anomalies of genito-urinary function: Enuresis; sexual irritability; impotence; sterility.

Anomalies of the instinct or appetite: Merycism; uncontrollable appetites for food, liquor, drugs, etc.

Diminished resistance to external influences and diseases.

Retardation of puberty.

**Psychical Stigmata.**—Insanity; idiocy; imbecility; feeble-mindedness; eccentricity; moral delinquency; sexual perversion.

#### DEAF-MUTISM.

Excluding the cases in which idiocy is present, which are not considered in this chapter, deaf-mutism may be due either to congenital or acquired conditions; the larger proportion of the cases belong in the latter class. When congenital, deaf-mutism may result from otitis, or periostitis of the temporal bone, encroaching upon the cavity of the middle ear, from ankylosis of the ossicles, from absence of the internal ear or any of its parts. There may also be colloid degeneration of the labyrinth. It may result from atrophy of the auditory nerve, and it may be due to a lesion of the brain. These congenital conditions are

often hereditary. Acquired deaf-mutism is most frequently the result of scarlet fever, and is due to otitis. The second important cause is cerebro-spinal meningitis, where it may be due to a lesion of the brain, the auditory nerve, or the ear. It occasionally follows mumps, diphtheria, measles, and other infectious diseases. It may result from repeated attacks of acute otitis associated with adenoid growths or chronic rhino-pharyngitis.

The younger the child at the time the deafness occurs the sooner the power of speech is lost. In most of the infectious diseases, if the attack occurs before the fifth year speech is lost. According to Love, total deafness is rare among deaf-mutes; hearing for speech is present to a useful degree in about twenty-five per cent of the cases, while hearing by cranial conduction exists in nearly all cases. Deaf-mutism should be suspected if a child not idiotic shows at the end of two years no signs of beginning to talk. A careful distinction should be made between deaf-mutism and idiocy resulting either from congenital conditions or acquired disease.

It is necessary that this condition be recognised as early as possible, in order that the child may have the advantages of proper training during his early years. The physician should insist upon the child being sent as early as the third, and certainly by the fourth year to an institution where it may be taught to speak.

The treatment is mainly prophylactic. The most important relates to the care of the ears in scarlet fever, and the removal of adenoid vegetations of the pharynx and other causes which produce attacks of acute or chronic otitis. For the condition itself education is the only thing to be considered.

## CHAPTER IV

### DISEASES OF THE SPINAL CORD.

#### MYELOMATOSES.

Myelomatoses of the spinal cord are associated with those of the brain, and bear a certain degree of resemblance to them. (1) The cord may be affected anywhere, as shown on the next page or may involve the whole of it. The local involvement may be only partial, the involvement may extend some distance in varying. The most important one is a local involvement of the spinal cord which may be a case of disseminated sclerosis. There may be a partial involvement of the cord, as in the case of a partial myeloma. There may be a complete involvement of the cord, as in the case of a complete myeloma, and is referred to as a complete myeloma. The spinal cord is

usually associated with other deformities. All of these malformations are extremely rare and of very little practical interest.

There remains to be mentioned the only one which is really important—*spina bifida*.

**Spina Bifida.**—This is a malformation of the vertebral canal with a protrusion of some part of its contents in the form of a fluid tumour. The tumour is elastic, compressible, usually increased by crying, and sometimes by pressure upon the anterior fontanel. The contained fluid is clear serum, resembling in all respects the cerebro-spinal fluid. It is one of the most frequent congenital deformities.

According to Humphrey, *spina bifida* is due to an early failure in development—in most cases before the cord is segmented from the epiblastic layer from which it is developed. Hence it remains adherent to the epiblastic covering, and the structures which should be formed between the cord and the skin are undeveloped. For this reason there is in the wall of the sac a fusion of the elements of the cord, nerves, meninges, vertebral arches, muscles, and integument. If the error in development occurs later, the cord and nerves may be attached to the sac, but not intimately fused with it; in still other cases the cord does not enter the sac at all. The malformation may occur before the central canal is closed; or, if closed, it may reopen from the accumulation of fluid. It is probable that the accumulation of fluid first occurs, and that this prevents the union of the parts of the vertebral arches.

Although the tumour is generally associated with a bifid spine, this is not necessarily the case. The protrusion may take place through the intervertebral notch or foramen, or there may be a fissure of the bodies of the vertebræ, and an anterior tumour projecting into the cavity of the thorax, abdomen, or pelvis—*spina bifida occulta*. The principal anatomical varieties are *meningocele*, *meningo-myelocele*, and *syringo-myelocele*.

**Meningocele.**—In this form there is a protrusion of the membranes only (Fig. 148). The accumulation of fluid is either in the arachnoid cavity or the subarachnoid space posterior to the cord. The opening of communication between the tumour and the spinal canal is small in this variety, usually being about one-twelfth to one-sixth of an inch in diameter. There may, however, be no communication. The skin is usually fully developed (Fig. 149). The tumour is frequently globular, sometimes pedunculated, and may attain a very large size, being as much as five or six inches in diam-



FIG. 148. — MENINGOCELE (partially diagrammatic). *A*, the membranes; *B*, the spinal cord; *C*, the integument. The accumulation of fluid is behind the cord, which does not enter the sac.

eter. This is because spontaneous rupture is not likely to occur, and the tumour does not become infected except by operative interference.

With such tumours patients may live to adult life. This variety is most frequently seen in the cervical region. It has the best chance of natural recovery, and in it operation gives the best results.



FIG. 149.—MENINGOCELE, IN A CHILD ONE YEAR OLD.

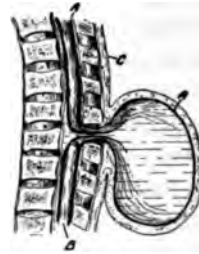


FIG. 150.—MENINGO-MYELOCELE (partially diagrammatic). A, the membranes; B, the cord; C, the integument. The accumulation of fluid is in front of the cord, the filaments of which are spread out, forming a part of the wall of the sac.

*Meningo-myelocoele.*—This is by far the most frequent variety of spina bifida, occurring in thirty-five of the fifty-seven cases reported by Demme. It is the form usually seen in the sacro-lumbar region. The accumulation of fluid takes place in the anterior subarachnoid space, less frequently in the anterior arachnoid cavity (Fig. 150). In this form the cord is contained in the sac, and usually forms a part of its wall. The tumour is smaller than the meningocele, the usual size being that of a mandarin orange. It is sessile, never pedunculated. As a rule it is only partly covered by skin, but has a central area, elliptical in shape, where there is only a thin, translucent membrane. This surface, which is known as the central cicatrix, is sometimes covered with granulations, and frequently ulcerates. The tumour often has a vertical furrow or a central umbilication, corresponding to the attachment of the cord on its inner surface. The usual relation of the parts is for the cord to run horizontally across the upper part of the tumour to the central cicatrix, with which it becomes blended, and from which again the nerves arise. These re-enter the canal at the lower part of the tumour, and are distributed below as usual. In other cases the cord joins the wall of the sac soon after its entrance, and its attenuated fibres are found spread out all over the sac, coming together again below and entering the spinal canal.

The following case, upon which I made an autopsy, is a good example of the common variety: The child died on the third day after birth from rupture of the sac. The tumour occupied the sacral region. The first sacral vertebra was normal, and beneath this the cord passed out of the spinal canal, terminating in the cauda equina soon after entering the sac, and continued back to the central cicatrix. Here nerve filaments blended with the other tissues in an indefinite structure, from which again, with tolerable distinctness, the nerve structures could be seen to pass over the wall of the sac and return to the canal. The afferent and efferent nerves and the part of the membranes they carried with them formed several septa, making a smaller separate sac within the larger one. The large sac was clearly a dilatation of the anterior subarachnoid space, and communicated freely with the same space in the cord above.

*Syringo-myelocoele.*—In this variety the accumulation of fluid is in the central canal of the cord, the lining of the sac being here the attenuated and atrophied cord elements. This is the rarest form of tumour, but the one most frequently associated with hydrocephalus, and consequently having the worst prognosis. It may be found in the dorsal or dorso-lumbar region as well as in the lumbo-sacral (Fig. 151).

With spina bifida other deformities are frequently associated, the most common being club-foot, hydrocephalus, more rarely encephalocoele or cerebral meningocele, and hare-lip. If hydrocephalus exists, there is in most cases a dilatation of the central canal of the cord and a direct communication between the tumour and the lateral ventricles of the brain. Pressure upon the anterior fontanel causes an increase in the size of the tumour, and conversely. Club-foot is usually double, most frequently talipes equinovarus. In a number of cases there is a history of some deformity in other members of the family. I once saw two successive children in the same family with spina bifida.

*Symptoms.*—The tumour in spina bifida is present at birth, and is most frequently lumbo-sacral. Paralysis is frequent in myelocoele and syringo-myelocoele, but is not seen in meningocele; its degree and its location depend upon the situation of the tumour and the extent to



FIG. 151.—SYRINGO-MYELOCELE OF THE MID-DORSAL REGION, IN A CHILD FOUR MONTHS OLD, WHO ALSO HAD HYDROCEPHALUS.



which the cord is involved. It is rare in cervical tumours, and most marked in those situated in the lumbo-sacral region. In the worst cases there is complete paraplegia, with paralysis of the bladder and rectum. If the tumour is sacro-lumbar or sacral, only the cauda equina is likely to be involved, and this but partially, so that the paralysis of the extremities is incomplete, and the bladder and rectum may escape.

In Fig. 152 is shown a very remarkable case of sacral spina bifida in a boy of five years, who came under observation for incontinence of



FIG. 152.—SACRAL SPINA BIFIDA.

fæces. The tumour was a little more to the left than to the right side, and had been overlooked. It had evidently pressed upon the lower branches of the sacral plexus, so as to affect the sphincter and the gluteal muscles of the left side. The atrophy was very marked, as shown in the illustration.

The natural course of spina bifida is to increase steadily in size; and if the tumour is covered by skin, its growth may be almost unlimited. It has been known to attain a circumference of twenty-two inches. If the integument is wanting, and the sac wall is very thin, rupture is pretty certain to take place, either spontaneously or by some accident, in the course of the first few months; death then results from convulsions owing to

the rapid draining away of the cerebro-spinal fluid, or from secondary infection. In a large number of cases death is due to marasmus dependent upon the associated conditions. Infection of the tumour may take place without rupture, the germs passing through the wall of the sac. If the opening communicating with the spinal canal is small, this infection may excite an inflammation limited to the wall of the sac, and result in a cure of the spina bifida, usually with sloughing. I have now under observation a girl ten years old in whom this occurred in infancy. The site of the former tumour is marked by a large dense cicatrix, and there still remains partial paralysis of the legs. If the opening into the spinal canal is large, inflammation of the sac is usually followed by spinal meningitis, which may extend upward and involve also the meninges of the brain.

*Prognosis.*—This depends chiefly upon the anatomical variety and the existence of complications. Simple meningocele, when covered by integument, gives the best prognosis, and complete recovery may occur. In meningo-myelocele, especially if complete paralysis exists, the prognosis is bad; and if there is hydrocephalus, the case is hopeless. In quite a number of cases in which cure of the spina bifida has followed operation, hydrocephalus has subsequently developed. Of fifty-seven cases reported by Demme, twenty-five were operated upon, with seven recoveries and fifteen deaths, while three were unimproved; of the thirty-two cases not operated upon, twenty-eight died within the first month, and not one lived over two years—the causes of death being marasmus, rupture of the sac, and meningitis.

*Diagnosis.*—It is usually easy to recognise spina bifida, but it is often difficult to distinguish between the different varieties. The absence of a palpable fissure in the spine, perfect translucency, and a pedunculated tumour, all point strongly to meningocele. Paralysis of the sphincters and lower extremities, umbilication of the centre of the tumour, a sessile tumour, a palpable bony fissure, and a large central cicatrix, point to meningo-myelocele. The coexistence of hydrocephalus points to syringo-myelocele.

*Treatment.*—In all cases the tumour should be protected from pressure, and care taken where it is not covered by integument, that the surface is kept absolutely clean and aseptic. It should be covered with some antiseptic powder and surrounded by a large pad of absorbent cotton, or a rubber ring-cushion. Complete paraplegia with involvement of the bladder and rectum, hydrocephalus, or extreme marasmus—all contraindicate operative interference. If these are absent, operation should be considered. The time of operation will depend somewhat upon the nature of the tumour. If it is covered by integument and growing slowly, it is well to wait until the child is at least six months old. In other cases delay is dangerous, because of the liability to spontaneous or accidental rupture.

The treatment by injection has now been entirely superseded by the operation of excision of the sac. For a description of this and the various plastic operations that have been proposed in connection with it the reader is referred to works upon operative surgery. In operating, it should not be forgotten that in the great proportion of the cases (ninety-five per cent, according to the London Clinical Society's Report, which, however, refers only to fatal cases) some part of the cord is in the sac. The cord is often present in tumours situated below the third lumbar vertebra, owing to its attachment to the sac.

Although recovery may follow operation, in a very large number of cases it is incomplete; some degree of paralysis, with atrophy, contractures, and deformities, remaining because of the implication of cord ele-

ments in the sac. In a considerable proportion of cases, hydrocephalus subsequently develops, as after similar operations upon cerebral meningocele.

#### SPINAL MENINGITIS.

In acute meningitis usually only the pia mater is involved. This rarely occurs alone, unless it is due to traumatism. It is most frequently associated with inflammation of the pia of the brain, and may occur either with the meningococcus or the tuberculous variety. A certain amount of acute inflammation of the pia mater accompanies most of the cases of acute myelitis.

Chronic spinal meningitis in children usually involves the dura only. Inflammation of the external layer (external pachymeningitis) is usually secondary to caries of the vertebræ. This is considered in the article on Compression-Myelitis.

**Symptoms.**—The symptoms of inflammation of the spinal membranes, no matter with what pathological condition it may be associated, are due to irritation of, or pressure upon, the cord or nerve roots. Those which are most common are: pain in the back, which is increased by movement, and usually by pressure upon the spinous processes; radiating pains following the course of the spinal nerves, felt in the extremities or in the trunk; rigidity of the spinal column due to spasm of the spinal muscles, or rigidity of the muscles of the extremities; and hyperæsthesia along the spine, which may be quite acute. When pressure upon the cord is added, there is paralysis or paresis, sometimes muscular atrophy and anæsthesia. Any of the above symptoms may be acute or chronic, according to the nature of the primary disease.

The diagnosis between spinal meningitis and myelitis is often not easy, for except in acute cases the two processes are usually associated; and in a given case it may be difficult to decide whether the lesion of the cord or of the membranes is the more important one. In meningitis, pain, tenderness, spasm, and irritative symptoms are generally more prominent, while loss of power and anæsthesia are usually partial. In myelitis the pain, tenderness, and other irritative symptoms are less marked, while paralysis and anæsthesia may be complete.

**Treatment.**—This relates first to the disease with which it is associated; in addition, counter-irritation by means of the Paquelin cautery, rest in bed, and in severe cases even immobilisation of the spine by a mechanical support. Iodide of potassium is often useful.

#### MYELITIS.

Myelitis is a rare disease in children, with the exception of two varieties which are discussed under separate heads, viz., compression-myelitis

and acute poliomyelitis. Otherwise myelitis usually results from injury, but it may occur as a complication of any of the acute infectious diseases, especially typhoid or scarlet fever, and diphtheria, and even as a primary disease, when it is attributed to exposure or cold, but when it is probably infectious. Chronic myelitis may be due to hereditary syphilis.

Myelitis usually occurs in children over ten years of age. In situation, it may be transverse, diffuse, or disseminated; the process may be acute, subacute, or chronic. The lesions and the symptoms are essentially the same as when the disease occurs in the adult.

**Symptoms.**—Myelitis usually comes on rather gradually, with only local symptoms; but the onset may be quite acute, with severe general symptoms—fever, pain, prostration, and localised or general convulsions. The local symptoms vary with the seat and the extent of the disease.

In transverse myelitis loss of power and anæsthesia are present below the level of the lesion; either of these may be partial or complete. At the level of the lesion there is a zone of hyperæsthesia and “girdle-pains.” All the reflexes below the seat of the lesion are exaggerated. Those at the level of the lesion are lost. There may be loss of control of the sphincters, bed-sores, degenerative changes in the paralysed muscles, contractures, and vaso-motor disturbances. The paralysed muscles may be rigid or flaccid, according to the seat and extent of the lesion.

When transverse myelitis is situated in the cervical region there are paralysis and anæsthesia of the arms, legs, and trunk. All the reflexes are exaggerated, and there is general rigidity of the paralysed muscles. There are incontinence of feces and retention of urine, followed by incontinence from overflow. The pupils are frequently contracted, and there may be optic neuritis. Atrophy, when present, usually affects the muscles of the arms, and indicates that the cord to a considerable extent is involved. There is great danger to life, owing to paralysis of the muscles of respiration.

When the seat of disease is the dorsal region, the symptoms are similar to those above described, with the exception that the arms escape, and that the eye-symptoms are usually wanting. This is the most favourable seat of the disease.

When the disease is situated in the lumbar region, in addition to paraplegia and anæsthesia of the legs, there is, from the beginning, incontinence of urine and feces. The knee reflexes are lost; the muscles atrophy, and usually give the reaction of degeneration. Bed-sores are frequent.

In diffuse myelitis the symptoms are a combination of the above groups. If a large part of the cord is involved, there are general paral-

ysis and anæsthesia, loss of reflexes, marked trophic disturbances, bed-sores, etc.

The course of myelitis is slow, and it usually progresses steadily from bad to worse. Death is due to exhaustion or complications—cystitis, bed-sores, or hypostatic pneumonia—or to some intercurrent disease. In a small proportion of the cases there may be partial recovery, but very rarely is this complete. The diagnosis is to be made from spinal meningitis, tumours, and hæmorrhage.

**Treatment.**—The treatment of the early stage consists in the use of ice to the spine, or counter-irritation by means of dry cups, mustard, or the Paquelin cautery. Later, the iodide of potassium should be given in all cases; improvement may follow its use, even when there is no suspicion of syphilis, but large doses are required, and for a long period. Electricity is contraindicated except in chronic cases, and then but little improvement is likely to result from its use. In these patients the most important thing is careful attention to cleanliness and to posture, in order to prevent bed-sores, cystitis, and pneumonia.

#### COMPRESSION-MYELITIS

*(Pressure-paralysis of the Spinal Cord; Pott's Paraplegia.)*

Compression-myelitis is sometimes traumatic, but usually follows caries of the spine. It most frequently complicates this disease when the cervical or upper dorsal vertebræ are involved, rarely when the lower half of the spinal column is affected. This difference is probably due to the smaller size of the spinal canal in its upper portion. According to Gibney, paraplegia is seen in fifty per cent of the cases of caries of the upper half of the spine. Essentially the same condition, so far as the cord is concerned, may result from tumours of the spinal cord, or from anything else causing pachymeningitis. These, however, are exceedingly rare in childhood.

**Lesions.**—In spinal caries there occurs as a result of tuberculous disease a softening of the bodies of the vertebræ, which fall together from the pressure due to the superincumbent weight of the body. This causes a backward projection known as the kyphosis, or angular deformity. The spinal canal is encroached upon by the remains of the vertebral bodies whose ligamentous attachments have been loosened, and also by inflammatory products the result of periostitis, and localised inflammation of the dura mater, chiefly of the external layer, but which sometimes affects the internal layer also. All these conditions lead to the production of a mass of inflammatory material, often containing tuberculous deposits, which is chiefly in front of the cord, but may surround it. The compression takes place slowly in most of the cases, from the gradual progress of the lesions mentioned. In a small number of cases there may be a

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sudden pressure from the slipping backward of one of the vertebral bodies.

In recent cases the cord at the seat of compression is a little smaller than normal. It is usually involved to the extent of from half an inch to two inches. Paraplegia may have existed when the changes found in the cord are very slight, and sometimes when no changes are visible to the naked eye. In more protracted and more severe cases, the cord is much smaller at the point of disease, and under the microscope shows the changes of interstitial myelitis (Gowers) with meningitis. In old cases there are degeneration of the nerve elements, atrophy, and sometimes disappearance of the ganglion cells, with more or less destruction of the nerve fibres; sometimes all distinction between the gray and white substance is lost. In addition to these marked changes at the point of pressure, there may be ascending or descending degeneration, as from other focal lesions. There is usually inflammation of the nerve roots, which have also suffered compression. It is in many cases surprising to see to what degree the cord may be compressed and still preserve its functions.

**Symptoms.**—In caries of the cervical region the symptoms of compression-myelitis not infrequently precede the deformity, and, in fact, the other objective symptoms of bone disease. The earliest symptoms of caries usually arise from irritation of the nerve roots, and consist of acute pains not often referred to the spine, but radiating to the different regions to which these nerves are distributed. They are felt in the neck, in the chest, in the epigastrium, and sometimes in the loins. Accompanying these pains, there is noticed a gradual weakness in the lower extremities, and sometimes also in the arms, according to the location of the disease. This may steadily increase for several weeks until there is complete paralysis. Other symptoms are then commonly present. There is usually some degree of anæsthesia, and there may be numbness, tingling, formication, and pain. The sphincters are not often involved. When the disease is in the upper half of the cord, there are rigidity of the extremities and great exaggeration of all the reflexes, with marked ankle-clonus. In the rare cases in which the lumbar enlargement is involved, there may be loss of reflexes, paralysis of the sphincters, and bed-sores.

The distribution of the paralysis will depend upon the point of compression. If this is in the cervical region, all four extremities will be paralysed; if in the dorsal region, only the legs. According to the extent of the secondary lesions in the cord, there may occur muscular atrophy and contractures. With disease in the upper cervical region, death may result from sudden pressure upon the cord, owing to a dislocation of the odontoid process; or there may be vomiting, pupillary symptoms, irritation of the phrenic nerve causing hiccough, or pressure causing paralysis of the diaphragm.

**Course and Prognosis.**—These depend much upon the treatment of the case. In many cases of paralysis occurring early in caries, complete recovery takes place in the course of a few weeks, sometimes in a few days, after the application of a proper mechanical support. In the cases which have been long neglected, or those in which the paralysis develops while proper mechanical treatment is being carried out, the chances are not so good. Gibney gives the following statistics of 133 cases under his personal observation: 31 proved fatal; 9 dying from myelitis, 14 from other diseases subsequent to recovery from the paralysis, and 6 from tuberculosis before complete recovery; 74 recovered from the paraplegia; 27 were recorded as improved or still under treatment. Relapses occurred in about fifteen per cent of the cases. The usual duration of the disease is from three months to two years. Recovery has often taken place in cases that have persisted for four or five years.

**Diagnosis.**—This is rarely difficult. Spinal caries should be suspected in every case when the symptoms point to transverse myelitis coming on without definite cause.

**Treatment.**—The indications are the removal of pressure and the fixation of the spine by a proper mechanical support. Other measures to be advised are the Paquelin cautery and the internal use of potassium iodide. From his very extensive experience, Gibney has more confidence in this drug than in all else except mechanical treatment. Large doses are required, often from sixty to ninety grains being given daily for months. The iodide should always be largely diluted. Patients should be kept scrupulously clean, and the position changed frequently to prevent the formation of bed-sores. Electricity is contraindicated. When the paralysis develops rapidly or occurs suddenly, relief may sometimes be obtained by the operation of laminectomy; but little is to be expected from this in the slow cases.

#### ACUTE POLIOMYELITIS.

*(Epidemic Poliomyelitis; Acute Infantile Paralysis.)*

There are few diseases regarding which our knowledge has increased so rapidly during recent years as acute poliomyelitis. The first great step in advance was made by Landsteiner and Popper, who, in the summer of 1909, succeeded in producing the disease in a monkey by intraperitoneal inoculation with the spinal cord of a patient dying of acute poliomyelitis. They were not successful in carrying the transmission further. But shortly after this Flexner and Lewis, using the intracranial method of inoculation, had no difficulty in reproducing the disease and transmitting it through an indefinite series of monkeys. No other animals seem to be susceptible. These observations, now many times repeated, have not only definitely established the infectious char-

acter of poliomyelitis, but have cleared up many doubtful points in its pathology.

Acute poliomyelitis is now regarded as a communicable, infectious disease which prevails both epidemically and sporadically. Although possibly its most characteristic lesions are in the anterior horns of the cord, any part of the central nervous system may be affected. The changes in the cord substance are preceded by lesions of the meninges. Although the name poliomyelitis is still retained, the scope of the term has been greatly widened.

This disease is characterised by an acute onset, with fever and usually other marked constitutional and nervous symptoms, from which there may be rapid recovery; but generally there follows early and extensive loss of power. After this there is usually seen a gradual improvement, and sometimes complete recovery. More often, however, there is left some permanent paralysis in certain groups of muscles, which undergo rapid and marked atrophy. Formerly, poliomyelitis was seen chiefly as a sporadic disease; but since the year 1905 epidemics have occurred with increasing frequency in various parts of the world, and especially in the United States since 1907. As it is most frequently seen in very young children, and as it is altogether the most common form of paralysis at this period, the old term of "acute infantile paralysis" is perhaps the most appropriate clinical designation.

**Etiology.**—Fully eighty per cent of the cases are seen in the first three years of life, the greatest incidence being in the second year. Persons of any age may be attacked, and in some epidemics the proportion of adult cases is quite large. Epidemics thus far observed have invariably occurred in the warm months; those in the United States, from July to October. Fully four-fifths of the sporadic cases also are seen during these same months.

Bartlett and myself<sup>1</sup> could find recorded, previous to 1905, but 34 epidemics or outbreaks of this disease. Most of these were small in extent, and the total number of cases reported in all was only 889.

The prevalence of poliomyelitis in an epidemic form really begins with the outbreaks in Sweden and Norway in 1905 and 1906. These were followed in 1907 by the epidemic occurring in New York City and vicinity which was the most extensive yet known; there were observed from July to October between 2,000 and 3,000 cases. Since that time poliomyelitis has been gradually spreading to nearly all parts of the United States and in its progress has distinctly followed lines of travel. It has been especially prevalent in Massachusetts and Rhode Island; in Minnesota, Wisconsin and Iowa, and in Pennsylvania. In the year

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<sup>1</sup> See American Journal of the Medical Sciences, May, 1908. Also Archives of Pediatrics, September, 1910.



1910 nearly 9,000 cases were reported in the United States. While the greatest number of outbreaks of this disease have been seen in this country, some of considerable size have occurred in Australia and in Germany. The same locality has rarely been the seat of an epidemic in two successive seasons.

The instances recorded, now numerous, of the occurrence of several successive cases in a family, strongly suggest that the disease is directly

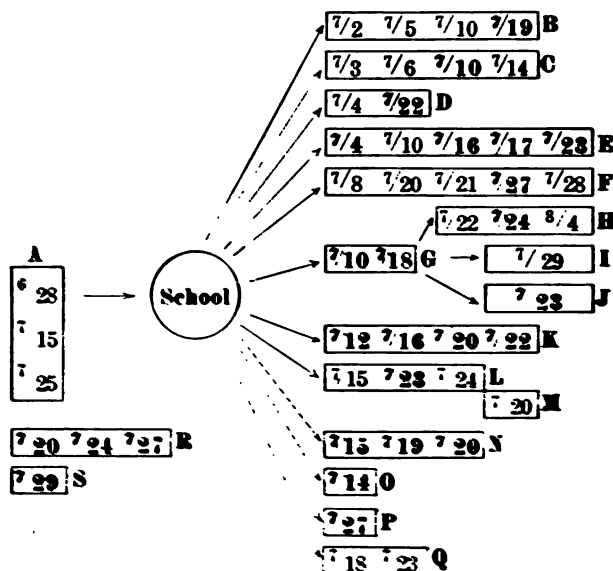


FIG. 153.—TRÖSTA EPIDEMIC OF POLIOMYELITIS. (After Wickman.) The epidemic was observed in a rural community of 500 persons in which 49 cases occurred in six weeks. The letters, A, B, C, etc., represent the different families in which cases occurred. The figures give month and day of onset of each case. Heavy figures indicate cases with paralysis; lighter figures, abortive cases. The relation of the families to the school is indicated by lines: in those connected by solid lines the children who attended school were first attacked; in those connected by dotted lines, some child attended the school, but the school child was not the first one attacked. The first case occurred in Family A, the child attending school while suffering from the early symptoms of the disease. In only two families, R and S, did cases occur in which there was no association with the school or with families whose children went to school.

communicable. In some epidemics, notably those of York, Nebraska, and Eastern Sweden (see Fig. 154), the source of contagion would seem to be a most probable one. There are a few instances in which the disease apparently was carried in a single person.

It is at present difficult to reconcile the facts indicating a high degree of communicability, such as has been witnessed in some epidemics, with what has been observed at other times and in other places. In many of the epidemics, when the disease has been of extensive extent, it has not been possible to trace any connection between the different cases. In the

majority of the families but one case occurred, although other children, quite as susceptible by reason of age, were closely exposed. For years we have received these affected children in the acute stage into hospital wards with other patients, and I have never yet known a secondary case to develop. Until recently, no effort whatever was made to isolate cases. At present we are able to recognise no differences between the sporadic and epidemic forms of the disease except in communicability and severity, which point to a greater virulence of the infection in the latter variety. Both etiologically and pathologically, the sporadic and epidemic forms seem to be identical. In all these respects the analogy to cerebro-spinal meningitis is a very close one.

The occurrence during epidemics of many unrelated cases strongly suggests some other mode of contracting the disease than directly from an infected person or a human carrier. The fact that epidemics are seen only during the summer and early autumn points in the same direction. Recent experiments of Flexner and Clark give some reasons for suspecting the house fly as a carrier. The question remains to be solved by future investigation.

The period of incubation of the experimental disease in monkeys varies from four to thirty-three days, the average being ten days. In man, also, it is variable, but in most instances the second case in a family has followed the first one within ten days.

*The Specific Virus.*—It belongs to the class of filtrable viruses, closely resembling, in many respects, the virus of rabies. It passes through the finest porcelain filter. It can not be seen with the highest power of the microscope, nor has it yet been cultivated outside the body. That it is a living organism is proven by the fact that it is destroyed by heat. It is present in largest quantity in the diseased nerve structures, particularly the spinal cord. In the earliest stages of the attack it is also found in the cerebro-spinal fluid, but disappears at about the time paralysis occurs. It exists to some degree in other tissues of the body, particularly the lymph nodes. The disease can be transmitted to animals regularly and with certainty only by inoculation with an affected spinal cord, in which the virus persists for months after the acute attack. Experiments indicate that the path of entrance may be the nasal mucous membrane, and at times the intestinal tract. Osgood and Lucas have shown that the virus persisted in the nasal mucous membrane of monkeys, in one instance for five months, in another for one and a half months, after the acute attack; which suggests that this may not only be an avenue of entrance, but a mode of elimination of the infection, and indicates that the duration of the infective period may at times be a very long one.


**Lesions.**—As a result of the investigations, particularly of Flexner and Lewis upon animals, and those of Harbitz and Scheel, Strauss, and others upon the disease in man, the pathology of acute poliomyelitis is

now well known. This knowledge has greatly aided our clinical understanding of the disease.

The virus of acute poliomyelitis first attacks the meninges, especially of the cord and medulla, setting up a cellular inflammation of the pia, which becomes infiltrated with small, round cells. These changes are most marked about the blood-vessels. Besides this the walls of the vessels themselves are infiltrated and their lumen narrowed. The lesion also affects the vessels entering the nerve structures. As a result of the vascular lesions anæmia, œdema, and hæmorrhages are present, sometimes small and circumscribed, sometimes quite diffuse and extensive. Thrombosis does not occur. But more important still are the degenerative changes in the nerve cells, the site and extent of which are determined by the vessels involved and the intensity of the changes in them. The lesions in the pons, medulla, and cerebrum, like those in the cord, are secondary to the vascular lesions.

The transient paralysis in cases that recover may be due to œdema or to temporary vascular obstruction from pressure outside the vessels causing a local anæmia. Permanent paralysis depends upon severe degeneration and actual destruction of ganglion cells; its extent, therefore, will vary with the number of the ganglion cells affected. Any part of the central nervous system may be affected, and the lesions are generally more extensive than the symptoms would lead one to expect. The gross appearances give but little idea of their severity. The process often involves nearly the whole length of the cord, being, however, generally most marked in the cervical and lumbar enlargements. The changes are chiefly in the gray matter of the anterior horns, and consist in acute degeneration of ganglion cells, usually marked and extensive. These cells in certain parts may disappear altogether, being replaced by leucocytes. The entire cord, however, may be involved. There is seen, but to a much less degree, infiltration with small round cells of the posterior horns, the columns of Clarke, and the white matter of the cord, everywhere closely related to the blood-vessels. There are regularly found changes in the spinal ganglia of a similar character to those described in the cord.

Lesions like those of the cord, though generally less marked, are seen in the pons, the medulla, the cerebellum, and even in the cerebral hemispheres. They are, as in the cord, especially related to the pia and the blood-vessels. There is seen acute destruction of ganglion cells and areas of cell infiltration with lymphocytes. The changes are especially marked about the nuclei of the cranial nerves, and in the gray matter about the fourth ventricle. In some cases the basal ganglia are also involved. Areas of infiltration, sometimes quite diffuse, may be seen in the cortex, with also some slight degeneration of ganglion cells.



Thus, in the severe and fatal cases there is present a diffuse inflammation of the entire cord and its membranes, also of the medulla, pons, and basal ganglia, with less marked changes in the cerebrum, always accompanied by changes in the pia. In the milder cases it is probable that the inflammatory changes are limited to the cord, though in some patients dying later from other causes Harbitz and Scheel discovered changes in the upper centres, though no symptoms pointing to them had been present. From this account of the lesions it would appear that we can no longer distinguish between the lesions of acute poliomyelitis, acute bulbar paralysis and acute poli-encephalitis inferior. They represent varying phases of one and the same disease. In recent acute cases no changes are usually found in the nerves except degeneration of bundles, corresponding to the degenerated areas in the cord, and probably secondary to them. Lesions in other organs are often present, the most frequent being broncho-pneumonia and acute parenchymatous degeneration of the liver and kidneys, similar to what is seen in other severe general infections. The thymus, the solitary follicles of the intestine, and the mesenteric glands may be much swollen.

In autopsies made upon cases of long standing the affected part of the cord, which is often only one lateral half, is smaller than normal. The general changes are those of a sclerotic character. The ganglion cells of the affected anterior horn have either disappeared altogether, or they are few in number and so shrunken as to be hardly recognisable. The white matter also is smaller than in the sound part of the cord. The anterior nerve roots are degenerated quite to the muscles. The affected muscles are atrophied, and in extreme cases there may be a complete disappearance of muscle fibres, their place being taken by adipose and fibrous tissue. In places where the lesion is less severe the fibres are small. The affected limb is shorter and the bones smaller than upon the sound side.

**Symptoms.**—The onset of a well marked attack of acute poliomyelitis is usually abrupt, being ushered in with fever, prostration, vomiting, rarely with convulsions. There may be diarrhoea, but more often there is obstinate constipation, and there may be retention of urine. Severe pains are usually present in the neck, the spine, and the extremities. There may be marked hyperæsthesia with so much rigidity of the neck and extremities as strongly to suggest cerebro-spinal meningitis. The mind is usually clear, but there may be active delirium or, rarely, drowsiness or stupor. The temperature usually ranges between 102° and 103.5° F. Such symptoms may continue for three or four days and then gradually subside and the patient recover without any paralysis having developed. Or there may be for a few days a general muscular weakness somewhat greater and lasting a little longer than would be expected in an illness of such severity. These are known as “abortive



*Extent and Distribution of the Primary Paralysis.*—In 560 sporadic cases in which this point was noted the distribution was as follows:

|  |            |
|--|------------|
| One lower extremity . . . . .  | 229 cases. |
| Both lower extremities . . . . .   | 176 "      |
| General paralysis of all extremities, and more or less of trunk. . . . . | 79 "       |
| One lower and one upper extremity . . . . .                              | 36 "       |
| Both lower extremities and one upper extremity . . . . .                 | 16 "       |
| One upper extremity alone . . . . .                                      | 14 "       |
| All other varieties . . . . .  | 10 "       |

In paralysis of the trunk, the abdominal muscles, the diaphragm, and other respiratory muscles are rarely affected. In combinations of an upper and a lower extremity, the limbs are more frequently affected upon opposite sides than upon the same side. The sphincters usually escape.

*Course of the Disease.*—After the constitutional symptoms have disappeared there is a period of from one to three weeks' duration in which little change is seen. This is followed by spontaneous improvement, which usually begins in the muscles last affected, and reaches its limit in about three months. The paralysis remaining after this time is likely to be permanent. By the end of six or eight weeks atrophy is present in the paralysed muscles. The affected limb is distinctly smaller than its fellow, this being quite apparent even in infants. Except in the early stage, sensory disturbances are absent; the knee-jerk is lost in paraplegic cases, and in those in which the extensors of the thigh are paralysed. There is arrested growth in the whole limb (Fig. 154). It becomes much smaller and shorter than its fellow. The great relaxation of the ligaments at the joints may allow subluxation, especially at the knee and at the shoulder. The circulation in the affected limb is poor; it is often blue and cold, but bed-sores are never seen.



FIG. 154.—AN OLD CASE OF INFANTILE SPINAL PARALYSIS OF THE ENTIRE LEFT LOWER EXTREMITY. Showing extreme atrophy of the thigh and leg, and a very characteristic deformity of the foot.

**Electrical Reactions.**—Very early in the disease the atrophied muscles begin to lose their power to respond to faradism. In the muscular groups which are to be permanently paralysed, the faradic response may be lost in a week. The muscles in which recovery is to take place often preserve a certain degree of contractility, although this is less than normal, and improves later. The response to the galvanic current may be increased for a few months, and then slowly fail as the muscular fibres themselves degenerate, and at the end of two or three years it may disappear altogether. The reaction of degeneration is present with great uniformity in the atrophied muscles, but in them alone.

**Residual Paralysis and Deformity.**—This is most frequently of one lower extremity. The extensors both of the thigh and of the leg are nearly always involved to a greater degree than the flexors. The muscles

most frequently affected are the anterior tibial group. Paralysis of one upper extremity rarely occurs alone, but is associated with paralysis of one or both lower extremities. Complete paralysis of an arm is very rare. Of single muscles, the one most frequently involved is the deltoid. From paralysis of the muscles of the trunk or shoulder of one side, lateral curvature may develop (Fig. 155).



FIG. 155.—AN OLD CASE OF INFANTILE SPINAL PARALYSIS OF THE LEFT ARM AND SHOULDER MUSCLES, WITH RESULTING LATERAL CURVATURE.

**Diagnosis.**—The recognition of acute poliomyelitis before the occurrence of paralysis is impossible except by lumbar puncture. If this is performed early, the cerebro-spinal fluid is found to be opalescent or slightly turbid, owing to the presence of many mononuclear cells. It may coagulate spontaneously. Noguchi's globulin test gives a positive reaction. By the time paralysis appears the cells have diminished greatly in number

and soon the fluid shows no changes by which it can be distinguished from the normal. Very exceptionally there has been seen early in the disease a marked turbidity and an excess of polymorphonuclear cells. Such cases are distinguished from meningitis by the absence of the characteristic organisms. The early symptoms—vomiting, constipation, or diarrhea and fever—usually lead to the opinion that this attack is only one of acute indigestion. When there are added muscular pains, general hyperæsthesia, rigidity, and high fever, cerebro-spinal meningitis is often suspected, and can be excluded only by lumbar puncture. Early cerebral symptoms, convulsions, stupor, etc., may closely simulate tuberculous

meningitis, and I have known doubt to exist for several days. Lumbar puncture and the examination of the fluid should settle the diagnosis.

The later manifestations of poliomyelitis are a flaccid type of paralysis with marked atrophy and with the characteristic electrical reactions, but without sensory symptoms. Seen late, poliomyelitis may be confounded with cerebral palsies, multiple neuritis, or the pseudo-paralysis of rickets. In cerebral palsies there is usually rigidity; there is no reaction of degeneration; other cerebral symptoms are commonly present, or there is a history of an onset with cerebral symptoms, and the atrophy is less marked. Multiple neuritis is rare in children except after diphtheria, and is more gradual in its onset. The type of paralysis and the electrical reactions may be the same as in poliomyelitis.

Certain birth palsies, especially those resulting from injuries received during delivery, may closely resemble poliomyelitis when the deltoid or shoulder group of muscles is involved. Without a clear history a differential diagnosis may be impossible.

The muscular weakness of rickets is general; there is no reaction of degeneration and no history of acute onset. Scurvy is distinguished by the very acute hyperæsthesia, by the swellings, and by hæmorrhages from the gums or other mucous membranes together with a history of improper feeding. The child refuses to move his legs only because of pain.

**Prognosis.**—It was once thought that few, if any, cases recovered perfectly, and on the other hand that there was very little danger to life. Wider observations which recent epidemics have made possible have shown that complete recovery may occur even in cases in which the onset is acute and early loss of power extensive. Such a result is, however, not the common one. The great majority of the cases have unfortunately some residual paralysis. Of the 1,659 cases occurring in epidemics collected by Bartlett and myself the mortality was twelve per cent. During the recent New York epidemic I saw personally four cases which ended fatally. The discrepancy between the mortality figures just mentioned and the opinion formerly held is possibly explained in part by the fact that in epidemics the more severe types of the disease are seen, but I believe is chiefly due to a failure to recognise the most severe forms, especially bulbar cases, as examples of this disease. Previous statistics have been gathered chiefly from neurological out-patient clinics, where the types which end fatally are seldom seen.

An important question in early prognosis is that which relates to the extent of the permanent paralysis. The significant symptoms are the amount of wasting and the electrical reactions. Muscles which in ten days have lost completely their faradic contractility are almost certain to waste rapidly and severely. The best indication of coming improvement is the return of faradic contractility. If this is completely lost for six months, recovery is doubtful; if for one year, improvement



in these muscles is not to be expected. If faradic contractility has never been lost, very great and early improvement in the paralysed muscles may be confidently predicted. After three months but little spontaneous improvement is to be looked for, and after two years none at all.

**Treatment.**—So little is as yet known of the mode of acquiring poliomyelitis that not much can be said regarding prophylactic measures. Inasmuch as the nasal mucous membrane is known to be at least a possible channel of elimination of the virus, it follows that all nasal discharges of patients should be carefully disinfected and destroyed. Persons in contact with active cases should use some antiseptic nasal spray. Strict quarantine of sporadic cases does not as yet seem to be necessary. In epidemics, however, immediate quarantine should be instituted and strictly maintained for at least a month. Further than this it is not now possible to make positive statements.

Even when recognised early, it is doubtful whether much can be done to limit the inflammation. The most important indication is to secure complete rest. Counter-irritation may be used over the spine by means of mustard or a Paquelin cautery, or an ice-bag may be employed; yet it is very doubtful if they have any influence upon the course of the disease. The results depend rather upon the severity of the attack than the treatment employed. The natural course of the disease is to be kept in mind, for the tendency is to overestimate the effect upon the paralysis of the drugs used in the early stage. In animals if hexamethylenamine (urotropin) is given simultaneously with or shortly after the injection of the virus in many instances no paralysis follows. Its curative effects in man have not yet been demonstrated. It should, however, be tried, and administered in full doses as soon as the diagnosis can be made. To a child of three years from twenty to twenty-five grains daily may be given in divided doses. It is doubtful whether drugs have any influence upon the paralysis after its full development.

After all acute symptoms have subsided, or at the end of two or three weeks, electricity may be used, but its curative effects have been very greatly overestimated. No amount of electrification can preserve muscles whose ganglion cells have completely disappeared. These muscles continue to waste and lose their faradic contractility, no matter how early electricity is begun nor how faithfully it is continued. Faradism may be used for such groups as respond to it; otherwise galvanism should be employed. The beneficial results from electricity are to be obtained chiefly in the first six months. Friction, massage, and manipulation are of undoubted value in improving the circulation and the nutrition of a limb, and should be faithfully continued twice a day for a long period.

**Mechanical Treatment.**—Mechanical appliances are useful to prevent deformity, also to furnish support to the limb in order to enable the child to walk. By such means many get about with tolerable comfort

for whom locomotion without apparatus is impossible without crutches. To overcome existing deformities in neglected cases, braces are employed in conjunction with myotomy or tenotomy of the various shortened tendons, excision of portions of elongated tendons, and the production of artificial ankylosis in cases of "flail joints." By these means the orthopædic surgeon is able to give a great deal of relief to these unfortunate and sometimes helpless patients.

#### TUMOURS OF THE SPINAL CORD.

Tumours of the cord are exceedingly rare in childhood, and almost unknown in infancy. The most common varieties seen in early life are glioma, sarcoma, and tuberculous tumours. Eisenschitz has reported a case of tuberculous tumour in the dorsal region occurring in a child of three and a half years. There was a similar growth in the cerebellum. The symptoms were essentially those of compression-myelitis.

In my service at the Babies' Hospital I had a case of glioma of the cord in a child only one year old, which was in many respects unique. The early symptoms were gradual paralysis of the upper extremities, to which were added later, stiffness of the neck, and finally immobility of the head—the position being that of typical cervical caries. During the sixteen days of observation there was high fever, from 101° to 104° F. There were no pupillary or vaso-motor symptoms. At the autopsy the cord was found to be the seat of a diffuse gliosis. In the cervical region there was marked enlargement, the cord being fully four times its natural size. A microscopical examination by Dr. C. A. Herter showed that the growth apparently began in the vicinity of the central canal, and that the gliomatous process involved the entire length of the cord.

A somewhat similar case has been reported by Miura in a boy of eight years.

The diagnosis of tumours of the spinal cord in infancy is practically impossible. In later childhood they are apt to be mistaken for Pott's disease, but the symptoms are the same as those seen in tumours of adult life.

#### SYRINGO-MYELIA.

Syringo-myelia, although a rare disease, is sometimes seen in early life. The term is applied to a condition in which there is a cavity in the cord the result of a pathological process, in contradistinction to the cases in which a cavity is the result of a malformation, or *hydromyelus*, although it is not infrequent for the two conditions to be associated. The pathological process which precedes the cavity formation is now thought to be, in most cases at least, an infiltration of the substance of the cord with gliomatous cells. The process is somewhat similar to that just described in the case of tumour of the spinal cord, with the excep-

tion that when it results in cavity formation it is slower. The infiltration in these cases usually begins near the central canal. It is followed by a degeneration and breaking down of the infiltrated areas, beginning at the centre. As the cavity forms it extends, and usually first invades the gray matter of the commissure, later the posterior gray horns, the posterior columns, or the anterior horns. The resulting cavity is usually irregular in shape, and may be very small, or may extend through a large part of the length of the cord. It is most frequently situated in the lower cervical and upper dorsal regions. It is filled with fluid, and surrounded by gliomatous tissue.

According to Starr, the essential symptoms are of three kinds: (1) There is progressive muscular atrophy, with paralysis of some or all the muscles of one limb, usually extending to the opposite limb and to the trunk, sometimes accompanied by the reaction of degeneration; (2) vasomotor and trophic disturbances in the affected limb, such as cyanosis, coldness, bullous eruptions, ulceration, abscesses, atrophy, and sometimes fragility of the bones and diminution of perspiration; (3) sensory disturbances, which are probably the most characteristic symptoms of the disease—there is loss of the sense of pain and of temperature in the atrophied part, while the sense of touch and of location may be preserved. The extent and distribution of these symptoms will of course depend upon the site of the disease.

The course of syringo-myelia is essentially chronic, the duration being usually several years; and although spontaneous arrest sometimes occurs the disease is in most cases steadily progressive.

The cause is unknown, and it is not influenced by any form of treatment.

#### FRIEDREICH'S ATAXIA.

This is a chronic disease of the spinal cord and medulla, which begins most frequently in childhood or about puberty. The lesion affects first the posterior columns, afterward the crossed pyramidal tracts, the direct cerebellar tracts in the lateral columns, and Clarke's columns in the gray matter of the cord. There is probably some disease of the medulla, the pons, and possibly of the cerebellum and the posterior nerve-roots. In advanced cases other parts of the cord may be involved. The disease is seen in certain families, often affecting several members in succession at about the same age. It occurs particularly in families where alcoholism, syphilis, and other nervous diseases are frequent.

Spencer, who has photographed upon this disease, gives the following as the characteristic symptoms: There is gradual loss of the lower extremities, but gradually extending to the upper extremities and the face. During the disease there is some weakness in the legs, especially in the anterior group of muscles. In the late stages this is marked

and accompanied by atrophy. The gait is peculiar, like that of ordinary ataxic patients, the difficulty in walking being due to the ataxia and not to the paresis. After a time there is produced a characteristic deformity of the foot—it is shortened, as if from pressure against the toes and the heel, the instep is high, and the extensor tendon of the great toe stands out prominently. This deformity is seen quite early in the disease. There is often lateral curvature of the spine. The knee-jerk is absent. Unprovoked and uncontrollable laughter is quite a characteristic symptom of the disease. The patient is unable to stand with his eyes closed. There are palpitation, occipital headache, and sometimes vertigo. In the later stages speech is slow and difficult, and the patient talks like one intoxicated. The expression of the face is vacant, and often nystagmus is present. There may be choreic movements. The symptoms steadily progress until the patient may be helpless, although the general health may remain good for years.

The disease is distinguished from locomotor ataxia by the absence of the "lightning pains," and of the bladder, rectal, or genital symptoms, the pupillary changes, the optic-nerve atrophy, and the trophic changes in the bones and joints. It is distinguished from cerebral tumour by the absence of headache, vomiting, and optic neuritis, and by its longer course. The progress of the disease is slow but steady. It may last from twenty to thirty years. It is incurable.

#### LANDRY'S PARALYSIS.

*(Acute Ascending Paralysis.)*

This rare disease is occasionally seen in early life. In regard to its etiology but little is definitely known, the usual causes assigned being the same as those of myelitis. Many cases diagnosed Landry's paralysis are undoubtedly examples of poliomyelitis. There is, however, no doubt of the existence of an acute ascending paralysis distinct from poliomyelitis.

It is characterised by a paralysis—sometimes preceded by general symptoms of malaise, fever, etc.—which begins in the legs and spreads rapidly to the muscles of the trunk and upper extremities; finally it may involve the neck, diaphragm, and muscles of articulation. The paralysis develops quite rapidly, often attaining its height in from twenty-four to forty-eight hours, sometimes even proving fatal within this time. In other cases it comes on gradually, and may be two or three weeks in reaching its maximum. There is dyspnoea from involvement of the muscles of respiration. The paralysed muscles are flaccid. There is hyperæsthesia, followed by partial or complete anæsthesia and loss of reflexes. There are no changes in the electrical reactions, no atrophy, no bed-sores, and usually no involvement of the sphincters. Occasionally the arms may be affected before the legs, and even the bulbar symptoms

may be the first noticed. Death is the most frequent termination, and in fatal cases the disease lasts from two days to a week. If recovery takes place, it is after two or three months of illness.

The indications for treatment are the same as in acute myelitis, for in the beginning the two diseases can not usually be distinguished from each other.

#### THE MUSCULAR ATROPHIES.

These cases may be broadly divided into two groups, following in the main the classification of Sachs: (1) those dependent upon disease of the spinal cord—the spinal atrophies; (2) those which are primarily diseases of the muscles themselves—the idiopathic atrophies.

In the group of atrophies of spinal origin belong (1) the “hand type” of Aran and Duchenne, which has been shown to be dependent upon a lesion of the spinal cord; (2) the “peroneal type” of Charcot, Marie, and Tooth, which as yet lacks positive pathological proof of its spinal origin, although its etiology, symptoms, and course leave but little doubt that it belongs in the same category with the hand type.

In the second (idiopathic) group are included (1) muscular pseudohypertrophy, and (2) the so-called “juvenile atrophy” of Erb, which is a much less frequent condition. These two varieties have the following features in common: There is progressive wasting, beginning early in childhood, and associated at some period with hypertrophy of certain muscles. There are no fibrillary contractions, no reaction of degeneration, and no lesions in the cord. From a pathological point of view these diseases might be more properly considered elsewhere, but they are so closely associated clinically with the spinal atrophies that it has seemed better to describe them in this connection.

**Progressive Muscular Atrophy of the Hand Type.**—This disease is characterised by a very slow but progressive wasting, which usually begins in the muscles of the ball of the thumb of one or both hands. Then the palmar group of muscles belonging to the little finger are affected, and later the interossei. When the wasting has reached a certain degree, there is produced a peculiar and characteristic deformity of the hand known as *main en griffe*, or “claw-hand.” Following these muscles, those of the forearm may be affected. At this point the disease is sometimes arrested, or the atrophy may extend to the muscles of the arm and shoulder, especially the deltoid, and finally to those of the back. Exceptionally, the atrophy begins in the muscles of the shoulder group or even in those of the leg. The wasting takes place very slowly, the muscles disappearing fibre by fibre, but the degree which may be reached is often extreme. The only other characteristic symptoms are fibrillary contractions in the muscles which are soon to atrophy. The patient is not conscious of them, but they are visible. The faradic

contractility is preserved just in proportion to the amount of muscle remaining. If the atrophy is complete, it is entirely lost.

The course of the disease is a very chronic one, covering many years. It is incurable. In rare cases the process may extend to the muscles of the tongue, affecting deglutition and articulation, and death may occur from interference with respiration; otherwise the disease does not tend to shorten life.

In this form of atrophy heredity is an important etiological factor. The disease may occur in children, but very often does not begin until after puberty. The lesion consists in an atrophy of the ganglion cells of the anterior horns of the spinal cord, followed by secondary degeneration of the anterior nerve-roots.

**Progressive Muscular Atrophy of the Peroneal Type.**—This is much less frequent than the variety just described. In this form, the first to waste are the anterior muscles of the leg, especially the extensor longus hallucis and extensor communis digitorum, afterward the peroneal group. The small muscles of the foot are next affected, and the disease may then go on to involve the muscles of the calf. At this point it may be arrested permanently, or for several years, after which the thigh muscles may waste like those of the leg. After many years the hands are in some cases involved as in the type previously described, and even the muscles of the forearm. As a rule, the supinator longus, the muscles of the shoulder, neck, trunk, and face, escape altogether. The atrophy is generally symmetrical, but not invariably so. The cutaneous reflexes are usually present. There is no pain. The reaction of degeneration is present in some of the muscles, and fibrillary contractions are frequent, but not always seen.

In this variety also the influence of heredity may often be traced. It is said that boys usually inherit the disease through the mother. Like the previous type, it begins late in childhood or not until after puberty.

As stated above, positive proof that this disease is due to a central lesion in the cord is as yet lacking. Analogy, however, leads to the belief that it depends upon changes in the ganglion cells of the anterior horns in the lumbar region, similar to those found in the cervical region in the hand type. The course of the disease is very chronic, and it is incurable. The resulting deformity resembles that seen after poliomyelitis, and may require the same mechanical treatment, with similar operations for relieving contractions.

**Muscular Pseudo-Hypertrophy** (*Pseudo-Hypertrophic Paralysis*).—This is the most frequent and best-known variety of the idiopathic atrophies. It is a disease of certain families, often three or four children being affected, the boys much more frequently than the girls. The symptoms as a rule come on early in childhood, nearly always before the tenth year. The earlier symptoms relate to a general weakness of the

lower extremities, which is accompanied by a marked increase in the size of certain muscular groups, usually those of the calves, but sometimes more of the thighs or the gluteal regions. Children walk late and unsteadily, and fall very easily. They have special difficulty in

rising from the floor and in mounting stairs. The method of rising is quite characteristic: the patient lifts his body until he touches the floor only with the hands and feet; then he proceeds to "climb up himself" by putting first one hand upon the knee, and then the other, gradually moving his hands higher and higher up the thighs until the erect position is attained. This is seen in most of the cases, but not in all.

The size attained by the calves is sometimes very great. Gowers mentions a case in which a boy of twelve had calves measuring fourteen and a half inches in circumference. The enlargement may affect almost any muscular group of the lower extremity. In the upper extremity, the *infra-spinatus* is most frequently enlarged, next the *supra-spinatus* and the *deltoid*. The *pectorals* and *latissimus dorsi* are never enlarged, but are generally markedly wasted. Most of these patients exhibit while standing a marked degree of lumbar lordosis, due to the weakness of the extensors of the hip. This is well shown in Fig. 156. The patient may be so weak upon his legs that the slightest touch will cause him to fall, even with his apparently immense muscular development. The small muscles are generally weaker than those which are enlarged.



FIG. 156.—MUSCULAR PSEUDO-HYPERTROPHY. Showing to a moderate degree the large calves and gluteal regions with a marked lordosis. (From a photograph by Dr. M. A. Starr.)

Later in the disease marked atrophy occurs with a corresponding weakness of all the affected groups, and the patient may be unable to walk or even stand. With the exception of the use of his hands, he may be absolutely helpless. The knee-jerk is at first normal, but gradually diminishes until it is finally lost. The electrical reactions are normal until marked wasting occurs, when there is a lessened response to fara-

dism and galvanism, but never the reaction of degeneration. There are no fibrillary contractions, and no sensory disturbances. The progress of the disease is generally slow, and sometimes irregular. It is often more rapid in early childhood, and slower after puberty. Many of these children, though apparently bright, are distinctly below the average for their ages.

The lesions are confined to the muscles. At autopsy they appear yellow, and microscopically there is found very marked atrophy of the muscle fibres, which in places have been almost entirely replaced by fat; there may be no trace of muscle left, the structure resembling adipose tissue. In other places there is an accumulation of fat between the atrophied muscle fibres, and a very great increase of the interstitial tissue.

The prognosis is grave, most patients dying before adult life is reached. The diagnosis is generally easy from the apparent hypertrophy and actual weakness of the muscular groups. The disease is incurable.

**The Juvenile Form of Muscular Atrophy.**—This is much less frequent than the form just described, but, like it, begins in childhood or early youth. It is characterised by progressive wasting of certain muscular groups, especially those about the shoulders and pelvis, and hypertrophy of others. Of the shoulder and upper extremity, the muscles affected are the pectorals, the trapezius, the latissimus dorsi, the serrati, the rhomboidei, the muscles of the upper arm, and the subscapularis. The deltoid, infra-spinatus and supra-spinatus for a long time escape, and may be hypertrophied. The hand and forearm are not involved. In the lower extremity, the muscles of the pelvis, thighs, and gluteal regions are affected, while those of the leg and foot escape. With this atrophy there may be associated a true or pseudo-hypertrophy of certain muscular groups. In this disease there are no fibrillary contractions, no reaction of degeneration, and no sensory disturbances. The course and result of this form are essentially the same as in the preceding variety. It is now generally regarded as closely allied to it in its pathology, the most important difference being that of localisation.

There has been described, chiefly by Landouzy and Déjerine, another form of atrophy known as the *infantile facial type*. In this, wasting begins in the muscles of the face; the lips are thickened, but all the rest of the facial muscles are markedly atrophied, giving a peculiar expression to the mouth known as "the tapir mouth." Later, the atrophy extends to the shoulders and arms, but does not involve the supra-spinatus or infra-spinatus, or the flexors of the hand and forearm. This is sometimes described as beginning in the shoulders, or even in the legs. The description therefore corresponds to the juvenile form of Erb, with the addition of facial symptoms, and it is probably a variety of the same disease.



## CONGENITAL MYATONIA.

*(Oppenheim's Disease.)*

This disease was first described by Oppenheim in 1900. It is a congenital condition and is usually noticed soon after birth. The striking characteristic is the loss of muscular power which always affects the lower extremities and these chiefly. The arms are less frequently and less seriously involved. In many instances the trunk and intercostal muscles are also affected, but the diaphragm, the muscles of deglutition and those supplied by the cranial nerves usually escape. The loss of power is apparently complete, but by close observation a few feeble contractions may sometimes be made out. The limbs are flaccid and flail-like. The electrical reactions are feeble but the reaction of degeneration is not present. All the reflexes are diminished and the patellar and Achilles reflexes absent. There are apparently no subjective symptoms. The infants are usually well nourished and may even be very fat. In those who live for several months or years the intelligence is apparently normal and control over the sphincters complete. The majority of the children suffering from this disease die during the first few months frequently of pneumonia, to which they are predisposed by reason of the condition of the respiratory muscles. Some few that survive beyond this period show a slow but progressive improvement. How long this may continue is as yet unknown.

The lesions are chiefly in the muscles, which may waste to fibrous cords or may largely be replaced by connective tissue and fat. In several of the cases the cells of the anterior horns of the cord have been reduced in number, sometimes almost absent, and the anterior nerve roots atrophic. The nervous lesion is regarded as a failure of development rather than a degeneration. It is believed by some to be the primary condition, the lack of muscular development being the result of deficient innervation. Little can be expected from any form of treatment.

## CHAPTER V

## DISEASES OF THE PERIPHERAL NERVES.

## MULTIPLE NEURITIS.

Under the term multiple neuritis are included those cases in which several nerves are affected in an inflammatory process, which may at times be general. This condition of the nerves is usually symmetrical and is accompanied by sensory changes.

**Etiology.**—The most frequent cause of neuritis in children is diphtheria, although it is caused also by other infectious diseases,

especially malaria, typhoid or scarlet fever, measles, and mumps. In diphtheria the inflammation is due to the direct action of the toxins upon the nerve structures, since it can be induced in animals by injecting toxins into the circulation. There is little doubt that in all infectious diseases the inflammation is excited in a similar way. The metallic poisons, lead and arsenic, are rarely the cause of multiple neuritis in early life, and the same is true of alcohol, although a marked case from this cause has come under my observation in a child only three years old.<sup>1</sup> Lastly, there are cases in which the cause assigned is simply exposure to cold—those classed as rheumatic.

**Lesions.**—Almost any nerves in the body may be affected, although the distribution varies somewhat with the cause of the disease. The musculo-spiral and the anterior tibial nerves are most frequently involved, but the inflammation may affect any of the spinal nerves, including the phrenic, and occasionally the cranial nerves, especially the pneumogastric, hypoglossal, oculomotor, and abducens. Several nerves in different parts of the body are usually affected, the lesion being in most cases symmetrical.

The affected nerve is sometimes red and swollen, owing to acute congestion and œdema or a sero-fibrinous exudation. In other cases the changes are almost entirely degenerative. The microscope shows the changes sometimes to be chiefly interstitial and sometimes chiefly parenchymatous. There is an exudation of cells into the sheath, between the sheath and the nerve fibres, and even between the nerve fibres themselves. The myeline breaks up into granules, and in places may completely dis-

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<sup>1</sup> This case was in many respects a remarkable one. The boy completely emptied a decanter containing twelve ounces of whisky, but almost immediately vomited the greater part of it. He soon after showed the symptoms of alcoholic intoxication, and in a few hours became comatose, in which condition he continued for twelve hours. After this he gradually lost power in his legs, and at the end of a week was unable to walk at all. He had convulsions, and after this there developed the usual symptoms of meningitis at the convexity, with which he was admitted to the Babies' Hospital, three weeks after drinking the whisky. The child was then unconscious and there was present incomplete paralysis, affecting all four extremities, with anæsthesia of the arms. The active inflammatory symptoms continued for six weeks longer, during which time there were repeated convulsions, continuous stupor, fever, gradually increasing deformities, marked atrophy, loss of reflexes, and great diminution in the faradic contractility of all the paralysed muscles; in the thighs, left leg, and abdominal muscles there were no responses to a strong current, but there was nowhere the reaction of degeneration. The child was at death's door for three or four weeks. Three months after the attack the first signs of improvement were observed in the cerebral symptoms. Shortly afterward he began to use his hands, and at the end of six weeks he was walking alone and talking freely. The improvement was very rapid, and eight weeks from the date of the first change for the better, and five months from the time of taking the whisky, he was as well as ever. The diagnosis was multiple alcoholic neuritis, with a convexity meningitis. (Fig. 157 is from a photograph taken while the symptoms were at their height.)

appear. The late changes are those of subacute or chronic degeneration of the nerve fibres.

With these changes in the nerves there are associated, in some cases, inflammatory and degenerative changes in the ganglion cells of the spinal cord, although they are much less severe than are the lesions in the nerves. However, they were once regarded as the explanation of some of these cases, particularly of diphtheritic paralysis.

**Symptoms.**—The onset of multiple neuritis is in most cases a gradual one, it being usually from two to four weeks before the paralysis reaches its height. Very exceptionally the onset may be abrupt, with fever, and marked paralysis in a few days. It is characteristic of this disease that both motor and sensory symptoms are present, and that they are the same in their distribution. The symptoms are usually symmetrical. There is first noticed a general weakness in the affected muscles, which slowly increases to complete paralysis. As the extensor groups of the hands and feet are apt to be affected, there are wrist-drop and foot-drop (Fig. 157). The paralysis may begin in the feet and hands,



FIG. 157.—ALCOHOLIC NEURITIS, SHOWING CHARACTERISTIC DROPPING OF THE FEET. This position of the lower extremities was maintained for over a month. Boy three years old.

and gradually extend until it involves not only the four extremities, but even the muscles of the trunk and the neck, although this is rare. The child may then be absolutely helpless, unable to sit up, or even to support his head. In such cases the head seems loosely attached to the body, and rolls about on the shoulders like a ball. Weakness of the spinal muscles leads to deformities (Fig. 158), which I have seen mistaken for Pott's disease, even by experienced observers. In most of the muscular groups the paralysis is incomplete. The symptoms which relate to the phrenic and the cranial nerves will be described with Diphtheritic Paralysis, for they are rarely seen in any other form. It is characteristic of multiple neuritis that the bladder and rectum escape.

The sensory symptoms are marked only in the early stage of the disease, while the paralysis is increasing; they improve so much more rapidly than the motor symptoms, that they may be altogether wanting at the time that the paralysis is at its height. In some cases they are so slight as to be overlooked. There is usually pain along the course of the affected nerves, which is sharp and neuralgic in character, and generally associated with acute tenderness of the nerve trunks and of the muscles. Often there is a general hyperæsthesia in the early part of the attack, followed by partial anæsthesia. The sensations of touch, pain, temperature, and the muscular sense are all about equally affected.

Ataxia is not uncommon, and may be a more striking symptom than the loss of power. All the reflexes are diminished or lost, especially the knee-jerk, as the legs are usually most affected. Sometimes, particularly after diphtheria, there is loss of the knee-jerk, when there is no other symptom of neuritis. In the severe cases muscular tremor is frequent.

Atrophy is a prominent symptom of neuritis, and it is evident early in the disease, often being quite as rapid as in poliomyelitis. The electrical reactions are altered—every grade of reduction in the responses being seen, from a slight diminution in the reaction to faradism to the complete reaction of degeneration. Vaso-motor symptoms, such as œdema of the affected parts, glossiness of the skin, etc., are often present. Deformities from muscular contraction occur early; they may be severe, and in some cases, permanent.

**Course and Prognosis.**—The usual course of the disease is for the symptoms gradually to increase for three or four weeks and then improve, sometimes rapidly, but more often slowly, the case usually going on to complete recovery in the course of a few months. Exceptionally the paralysis may be permanent. The sensory symptoms always disappear before the motor ones. Multiple neuritis may prove fatal, from paralysis of the heart or the muscles of respiration, or death may be due



FIG. 158. — MULTIPLE NEURITIS AFTER DIPHTHERIA IN A CHILD FOUR YEARS OLD. The position of the head and spine is due to partial paralysis of the trunk and neck. The legs were also affected.

to asphyxia from the entrance of food or foreign bodies into the air passages, owing to anæsthesia of the epiglottis and paralysis of the muscles of deglutition. Death sometimes follows from complications, especially pneumonia. The electrical reactions are of much prognostic value in regard to the persistence of the paralysis. If the reaction of degeneration is present the paralysis is certain to last many months, and some muscles are sure to be permanently affected. Where there is simply a diminution in the faradic responses, even though accompanied by marked atrophy, complete recovery may be expected, although it is often slow.

**Diagnosis.**—The diagnostic features of multiple neuritis are the combination of motor and sensory symptoms with the same distribution, the occurrence of atrophy, and the diminution in the electrical responses, even the reaction of degeneration. The gradual onset and the widespread distribution of the paralysis are also characteristic. If all four extremities are paralysed, it is altogether the probable disease; and if to this is added paralysis of the neck and spinal muscles, the diagnosis is almost certain. The facts that the paralysis is often incomplete, and that it involves parts distant from each other, are also important. Neuritis may be mistaken for poliomyelitis, for Landry's paralysis, or for Pott's paraplegia; an important diagnostic point from the last mentioned is the condition of the reflexes—being greatly exaggerated in Pott's paraplegia, while they are diminished or lost in multiple neuritis.

**Treatment.**—As this disease tends in the great majority of cases to spontaneous recovery, it is difficult to estimate the value of any method of treatment. Causes, such as lead, arsenic, alcohol, and malaria, are to be sought and removed as the first step. During the acute stage the pain may be so severe as to require relief, which is best accomplished by the application of heat. In using counter-irritation care is necessary, and such active measures as cauterisation should not be employed, for troublesome ulceration may follow. After the acute stage has passed, or at the end of three or four weeks, electricity should be begun, faradism being used if the muscles respond to a moderate current, otherwise galvanism. This should be continued daily until recovery. Strychnine is much used in these cases, but it is doubtful whether it has any specific influence, although as a tonic it is valuable. Other tonics, such as iron, quinine, and cod-liver oil, should also be given. Massage is also beneficial. The spinal treatment of cardiac and respiratory paralysis will be discussed in the following article.

#### NEURITIC PARALYSIS

This is not one of the most frequent variety of multiple neuritis, but it has some peculiarities which make a separate consideration of it desirable.

**Frequency.**—According to the statistics of various observers, paralysis, including all varieties, occurs after diphtheria in from 5 to 15 per cent of the cases. Sanné gives 11 per cent in 2,448 cases; Lennox Browne, 14 per cent in 1,000 cases (in neither of these groups did the patients receive antitoxine); the Report of the Collective Investigation by the American Pædiatric Society, 9.7 per cent of 3,384 cases which were treated by antitoxine.

It is difficult to state to what degree the frequency of paralytic sequelæ after diphtheria is affected by the antitoxine treatment. The figures above given might indicate that the protective power of the serum over the nervous tissues is not so great as over others, and that unless administered very early it has little or no influence. The more probable explanation of the frequency with which paralysis is seen after antitoxine treatment is that patients now live long enough to develop paralysis, when without antitoxine the same patients would have died during the early stage of the disease.

Being one of the direct effects of the diphtheria toxine, neuritis is much more likely to follow severe than mild cases; however, its occurrence after some very mild attacks shows how great is the susceptibility of the nervous tissues to the action of this poison. Sometimes the throat symptoms have been entirely overlooked, and the development of paralysis has been the first thing to arouse a suspicion of previous diphtheria.

**Time of Occurrence.**—During the second week, and sometimes even during the latter part of the first week, the early paralysis occurs, usually affecting the palate. The most frequent and most characteristic paralysis—that affecting the throat, eyes, extremities, and respiration—begins at a later period, usually from one to three weeks after the throat has cleared off, and sometimes even later than this.

**Extent and Distribution of the Paralysis.**—Ross gives the following statistics of 171 collected cases of diphtheritic paralysis: Palate affected in 128; eyes in 77, in 54 of which the muscles of accommodation were involved; lower extremities in 113; upper extremities in 60; trunk or neck in 58; muscles of respiration in 33. I do not think this represents the actual frequency of the different varieties so truly as do the American Pædiatric Society's figures, which give the forms of paralysis noted in a series of cases collected for another purpose. In 328 cases of paralysis, the variety was mentioned in 189; in 124 the throat was affected; in 22 the extremities; in 11 the eyes; in 5 the muscles of respiration; in 32 the heart; in 1 the neck only; in 8 the paralysis was "general."

**Symptoms.**—In the great majority of cases the throat is affected, and usually the paralysis is first noticed there. It may involve the palate alone, or the muscles of the pharynx or larynx in addition. The muscles of the extremities or of the eye are often next attacked. In severe cases

there may also be involved the muscles of the trunk and neck, and sometimes the diaphragm. It is this which distinguishes diphtheritic paralysis from other forms of multiple neuritis. Whatever the extent or situation of the paralysis, the knee-jerk is nearly always lost. The symptoms in the extremities and the trunk do not differ from those of multiple neuritis from other causes. The throat paralysis shows itself by a nasal voice and by regurgitation of fluids through the nose, sometimes by difficulty in swallowing or by the entrance of food into the larynx, owing to anæsthesia of the epiglottis and paralysis of the muscles of deglutition. There may be difficulty in protruding the tongue or in articulation. Facial paralysis is very rare. On the part of the eye there is most frequently seen inability to read, owing to paralysis of the muscles of accommodation; there may be dilatation of the pupils, rarely strabismus or ptosis.

Respiratory paralysis may be due to involvement of the phrenic or the intercostal nerves, more frequently the former. Extensive paralysis of other parts—the throat, extremities, or trunk—usually precedes. The first warning is generally in the form of occasional attacks of dyspnoea, sometimes accompanied by cough. Gradually these attacks increase in frequency and severity. The voice is reduced to a whisper. As the diaphragm is usually affected, the breathing is entirely thoracic. The respiratory movements are rapid, but irregular, shallow, and ineffectual. There is cyanosis, also great subjective as well as objective dyspnoea. The anxiety, distress, and apprehension of the patient are sometimes terrible. There is a constant dread of impending suffocation, and the respiratory movements are continued only by the patient's constant efforts, otherwise they would cease altogether. After a few hours these severe symptoms may subside, to return after a short respite. There may be several such attacks during two or three days, in each of which death seems imminent. Unfortunately, this is the most frequent termination. Of thirty-three such cases collected by Ross, only eight recovered. Associated with these respiratory symptoms others may be present. There may be attacks of abdominal pain, vomiting, and disturbance of the heart's action—usually an irregular or intermittent pulse, which may be either unnaturally slow or very rapid. In many cases the heart continues to beat normally, even though the respiration is so much disturbed.

The premonitory symptoms of cardiac paralysis are an irregular or intermittent pulse, often slow, but becoming very rapid from even the slightest exertion. It is always weak and compressible. The first sound of the heart is feeble and may be reduplicated. As the symptoms increase there are marked pallor, coldness of the extremities, great restlessness, anxiety, precordial distress, and perhaps orthopnoea. Within twenty-four hours from the beginning of such symptoms death usually occurs. In other cases it may come suddenly without any warning, or with a warning so slight as to be overlooked. At such times it often follows

some muscular exertion, such as getting out of bed, walking across the room, or so slight an effort as sitting up suddenly in bed. Fits of temper or other excitement have at times produced it. It is by no means certain that cardiac paralysis is due to a lesion of the cardiac nerves. Toxic myocarditis appears to be a more important factor in producing the fatal result.

Death in diphtheritic paralysis is usually due either to cardiac or respiratory paralysis. Of 171 cases of all varieties collected by Ross, forty-five were fatal.

**Treatment.**—Cases of paralysis of the trunk or extremities are to be managed like others of multiple neuritis. In severe forms of throat paralysis feeding by a stomach tube should be employed, on account of the danger of the entrance of food into the air passages. It must in most cases be continued for several days. The tube may be passed either through the mouth or the nose.

The great mortality attending paralysis of the heart and respiration shows how unsuccessful is treatment in most of the cases; still, no doubt there are instances where life may be saved by judicious treatment. In cases of threatened heart paralysis, the drug most to be depended upon is morphine, hypodermically; this should be used every two or three hours in sufficient doses to keep the patient under its influence while threatening symptoms are present. The patient should be kept absolutely quiet, not even being allowed to turn in bed. In respiratory paralysis the general reliance is upon strychnine used hypodermically in full doses, and faradisation of the respiratory muscles, particularly the diaphragm.

#### FACIAL PARALYSIS.

Peripheral paralysis of the face occurring as a result of injury inflicted during delivery has already been described. There remain to be considered here cases which arise from causes that operate at a later period. The facial nerve may be affected in any one of three situations—after its exit from the cranium, in the bony canal, and within the cranium.

In the first situation, the principal cause of neuritis is exposure to cold (the “rheumatic” cases), but it occasionally occurs as a complication of mumps and disease of the lymph glands of this region. The nerve is affected just after it has escaped from the stylo-mastoid foramen, and all the branches given off beyond its exit are involved. There is paralysis of the muscles of the forehead, those about the eye, cheek, nose, and mouth. The affected side of the face is smooth, there is inability to wrinkle the forehead, contract the eyebrows, close the eye completely, raise the nostril, whistle, or blow. The mouth is drawn to the healthy side (Fig. 159). If the paralysis is complete, there may be difficulty in drinking or in articulation. In partial paralysis the symptoms may



not be noticeable while the face is at rest. There are no sensory symptoms. The electrical reactions resemble those of other forms of neuritis; there is diminution in the response to the faradic current, which is more

or less marked according to the severity of the lesion, and there may be the reaction of degeneration.

In the bony canal, the facial nerve is usually inflamed as a result of disease of the ear. In children this is much more frequent than from the other causes just mentioned. While it occasionally occurs with acute otitis, it generally accompanies the chronic form with caries of the petrous bone which is very often tuberculous. In addition to the paralysis there is present or there is a history of a discharge from the ear, and generally there is some deafness upon the side affected. The facial symptoms are usually the same as in the cases first described.



FIG. 159. — FACIAL PARALYSIS OF RIGHT SIDE FROM MIDDLE-EAR DISEASE IN A CHILD TWO AND A HALF YEARS OLD.

However, when the nerve is affected between the stapedius and the geniculate ganglion, there is a disturbance of the sense of taste, and of the secretion of saliva. Facial paralysis also occurs as a result of injury to the nerve during the mastoid operation.

At the base of the brain the trunk of the nerve may be involved in cerebral tumour, basilar meningitis, and in fracture of the skull. In any of these conditions the auditory nerve also is likely to be affected.

**Prognosis.**—The result is greatly modified by the causes in the different cases. In those which are due to cold, spontaneous recovery usually occurs in the course of a few weeks or months. In those depending upon disease of the ear, the outlook is not so favourable, and though there may be improvement, it is not rare for some paralysis to be permanent. In the third group of cases, facial paralysis is only one of the symptoms, and the result depends entirely upon the nature of the cause.

**Diagnosis.**—Facial paralysis is easily recognised. It is important to separate the peripheral paralysis from that due to a lesion above the pons, as in cases of ordinary hemiplegia. In the latter group only the lower half of the face is affected, the muscles of the forehead and those about the eye escaping, and the electrical reactions are unchanged.

**Treatment.**—This is essentially the same as in other cases of neuritis. In cases due to ear disease the primary lesion should receive appropriate treatment.

## SECTION VIII.

### DISEASES OF THE BLOOD, LYMPH NODES, SPLEEN, BONES, AND JOINTS.

#### CHAPTER I.

##### *DISEASES OF THE BLOOD.*

THERE are several particulars in which the blood of infancy and early childhood differs from that of older persons.

**Specific Gravity.**—This has no constant relation to the number of white or red corpuscles, but varies with the amount of hæmoglobin. The highest specific gravity is seen in the blood of the newly born. During the first two weeks of life it sinks rapidly to its lowest point, where it remains until about the end of the second year; after this time it rises gradually until about puberty. The average specific gravity during childhood is 1.050 to 1.055.

**Hæmoglobin.**—The percentage of hæmoglobin is highest in the blood of the newly born, and falls rapidly during the first few days after birth. Throughout childhood it is considerably lower than in adult life. The hæmoglobin is lowest between the third month and the second year; after the second year it gradually increases up to puberty. The usual range in young children, as measured by the adult standard, is between sixty-five and eighty-five per cent, sixty-five per cent being a low limit in healthy children.

**Red Corpuscles.**—The number of red corpuscles is highest in the newly born. At this time it is from 4,350,000 to 6,500,000 in each cubic millimetre. In infancy it is from 4,000,000 to 5,500,000; in later childhood, from 4,000,000 to 4,500,000 (Hayem). In size a much greater variation is seen in the red cells of the newly born than in those of older children and adults. In the blood of the fœtus there are present nucleated red corpuscles or normoblasts (Plate XV, A). These diminish in number toward the end of pregnancy. They are always found in the blood of premature infants, but in infants born at term they are seen only in small numbers and disappear after a few days. In later infancy their presence is always pathological.

**Normal White Cells.**—The following varieties are found in health:

1. *Lymphocytes.*—These are small cells about the size of a red blood cell. The protoplasm is small in amount, forming merely a narrow rim

about the nucleus; it stains with basic dyes rather more deeply than does the nucleus. The nucleus is relatively large, is centrally situated, and shows at times one or two nucleoli. The protoplasm may have a reticular structure. These cells form in adults from twenty-two to twenty-five per cent of the white corpuscles, but in children they are often as high as fifty or sixty per cent (Plate XV, B, 10).

2. *Large Mononuclear Leucocytes and Transitional Forms.*—These cells are two or three times the size of ordinary red cells (Plate XV, D, 10). The oval nucleus is not so centrally situated as in the lymphocytes, and stains feebly but rather more deeply than the protoplasm, which is poorly stained by basic dyes. The protoplasm is homogeneous and relatively large in amount.

The transitional forms occasionally contain a few feebly staining neutrophilic granules; their nuclei are bent or curved and stain more deeply.

3. *Polymorphonuclear Neutrophiles.*—These are smaller than the large leucocytes (Plate XV, B and C, 8). The nucleus consists of three to four parts, usually connected by narrower portions, and stains darkly. The protoplasm stains with acid dyes and shows a great number of granules which stain only with neutral dyes. In adults these cells form about seventy per cent of the white cells; but in children they are less numerous, the increase in the lymphocytes being at the expense of the neutrophiles.

4. *Eosinophiles.*—These are about the same size as the neutrophiles (Plate XV, C, 9); they have deeply staining nuclei, usually divided into two parts. The protoplasm has many large granules that stain deeply with acid dyes, and often a narrow outer layer stains more deeply than the rest. They form from two to four per cent of the total number of white cells.

5. *Mast Cells.*—They are only occasionally found, their proportion being about 0.5 per cent of the white cells; they are mononuclear or polymorphonuclear cells whose granules stain only with basic dyes, not at all with tri-acid; often they are metachromatic (Plate XV, C, 12).

**Pathological White Cells.**—Of these there are two principal forms:

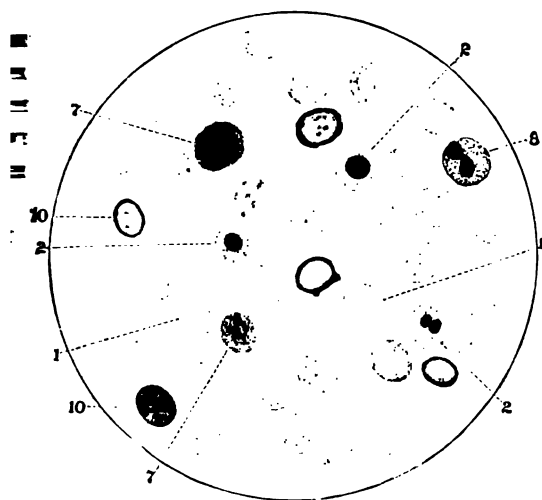
1. *Myelocytes.*—They have neutrophilic granules and a single rounded nucleus (Plate XV, C, 11). Ehrlich's myelocytes differ from those of Cornil in that the cells as a whole are smaller, the nuclei are more centrally situated and stain more intensely.

2. *Mononuclear Eosinophiles.*—These resemble the polynuclear eosinophiles, except for the round undivided nucleus.

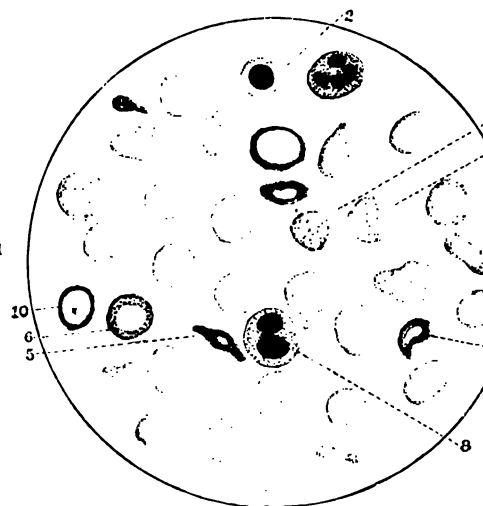
Pathologically, any of the leucocytes may undergo acute or chronic degeneration, with swelling and fragmentation, nuclear changes, hydropic degeneration, etc.

*The number of leucocytes* in the blood of the newly born, according to Rieder, is at birth from 14,200 to 27,400 per cubic millimetre; from

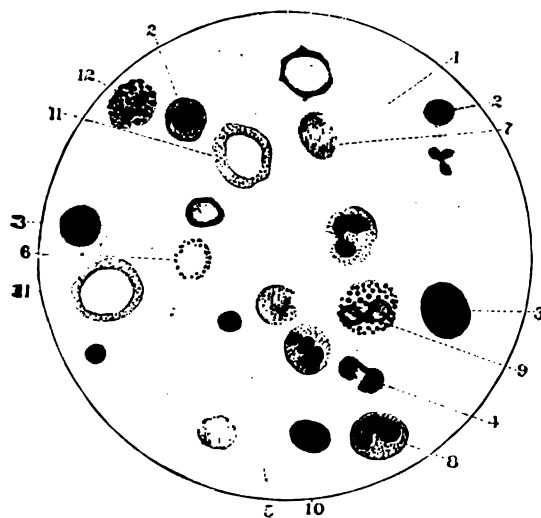
# PLATE XV.



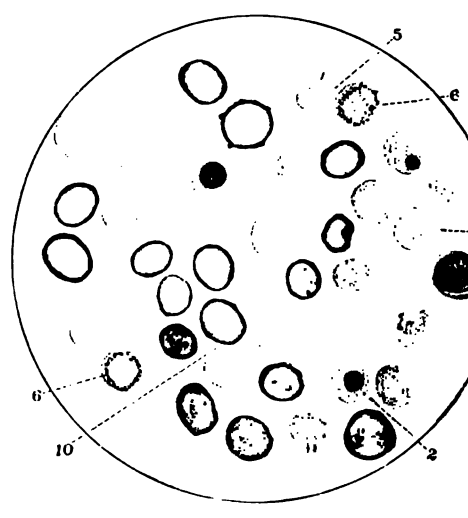
A.



B.



C.



D.

*Drawn by Dr. F. C. Wood.*

**A. BLOOD OF AN EIGHT-MONTHS' FETUS.**

**C. VON JAKSCH'S ANÆMIA.**

1. Red cells, normal.
2. Red cells, normoblasts.
3. Red cells, megaloblasts.
4. Red cells, showing mitosis.
5. Red cells, poikilocytes.
6. Red cells, granular degeneration.

**B. SIMPLE ANÆMIA.**

**D. ACUTE LYMPHATIC LEUKÆMIA.**

7. Red cells, polychromatophilia.
8. White cells, polynuclear neutrophiles.
9. White cells, eosinophiles.
10. White cells, lymphocytes.
11. White cells, myelocytes.
12. White cells, mast cells.



the second to the fourth day, from 8,700 to 12,400; after the fourth day, from 12,400 to 11,800. The variations in infancy are from 9,000 to 14,000, and in later childhood from 6,000 to 12,000.

#### LEUCOCYTOSIS.

By leucocytosis is meant an increase in the white corpuscles of the blood. This may relate to all or any of the varieties; although it is chiefly of the polymorphonuclear neutrophiles, there is seen in children a greater tendency than in adults to an increase in the lymphocytes.

It is customary to distinguish between physiological leucocytosis, such as that which follows a full meal, exercise, cold baths, or that which occurs in the newly-born infant, and pathological leucocytosis which occurs principally in inflammatory and toxic conditions, but may be seen also in malignant disease and after serious hæmorrhage. Digestive leucocytosis, that which occurs after feeding, is especially pronounced in children, the increase frequently amounting to thirty-three per cent of the total number of leucocytes present. Leucocytosis of the newly born has already been mentioned.

Leucocytosis is present in a great variety of pathological conditions. In many of them its significance is not yet fully understood; further study of it has not fulfilled the expectations of those who had hoped to obtain from it exact information regarding many pathological processes.

The form of leucocytosis which is chiefly important in children is the inflammatory. This is most marked in acute pneumonia, diphtheria, and in inflammations attended by the formation of pus. Leucocytosis is also frequently present in scarlet fever, erysipelas, acute rheumatism, septic and cerebro-spinal meningitis, and in many other conditions. Of the purulent inflammations, it is especially important in appendicitis, peritonitis, empyema, pyæmia, osteo-myelitis, and all acute abscesses. In the conditions above mentioned the increase is chiefly or exclusively in the polymorphonuclear neutrophiles.

There are other conditions, especially pertussis, hereditary syphilis, and certain diseases of the spleen, in which the proportion of the lymphocytes may be increased.

The eosinophiles are increased in leukæmia, in asthma, with intestinal parasites especially tapeworm, hookworm and trichinæ, and in some forms of chronic skin disease.

As a rule, leucocytosis is absent in typhoid fever, measles, malaria, influenza, and in tuberculous inflammations. It is wanting in the usual forms of gastro-enteritis of infants although it is marked in the type known as "Finkelstein's food intoxication."

Leucocytosis may be regarded as the reaction of the organism to the toxins in the blood elaborated by the bacteria concerned in the inflammation or infection, or to the bacteria themselves. It thus depends

upon two factors: the severity of the infection, and the amount of resistance of the individual, the latter being the more important. A severe infection with a high degree of resistance produces the most marked leucocytosis, while with very feeble resistance and the same infection the leucocytosis is slight or possibly absent. The degree of leucocytosis is also influenced by the nature of the inflammatory process, it being less marked in serous inflammations and more pronounced in suppurative processes. In inflammations it is usually greatest during the active stage of exudation.

**The Diagnostic Value of Leucocytosis.**—The following are the principal diseases in which a leucocyte count may be of clinical assistance:

*Appendicitis.*—A leucocytosis usually exists from the beginning; a marked or steadily increasing leucocytosis is to be regarded as an important indication for operation.

*Pneumonia.*—A marked leucocytosis is a characteristic feature of this disease; the exceptions are very mild cases or very severe infections with little or no reaction. The increase begins shortly after the onset and continues during the stage of exudation, generally reaching its maximum shortly before the crisis, when it declines rapidly. The usual number of white cells in an average case of pneumonia in a young child is from 15,000 to 30,000, but it is not rare for the count to run up to 40,000 or even 50,000. I have seen it over 100,000. The absence of leucocytosis in a strong child who is acutely ill is always strong presumptive evidence against pneumonia. A well-marked leucocytosis is of much value in differentiating pneumonia from typhoid fever, tuberculosis, influenza, or bronchitis.

*Empyema.*—A rapid increase in the leucocytes in the active stage of a pneumonia or after defervescence, in the absence of physical signs pointing to an extension of the pneumonic process, almost invariably indicates empyema. After the acute stage of empyema has passed there may be no leucocytosis whatever.

*Typhoid Fever.*—Leucocytosis is regularly absent in typhoid; its presence in an undoubted case indicates complications.

*Pertussis.*—A leucocytosis with a high proportion of lymphocytes is of considerable value in the diagnosis of this disease; it is more fully considered in the special chapter devoted to Pertussis.

*Meningitis.*—As a rule, a marked leucocytosis is present in all forms of acute meningitis except the tuberculous. In the latter variety it is not constant, and if present is generally less marked than in the other forms.

*Tuberculosis.*—Leucocytosis is regularly absent in unmixed tuberculous infections. It is occasionally found in tuberculous meningitis.

In surgical diseases the presence of leucocytosis is considered a reliable guide as to the existence of acute suppuration, although not always

as to its degree. An increasing leucocytosis is usually an indication for operative interference in cases where operation is admissible. This applies particularly to appendicitis.

#### SIMPLE ANÆMIA.

This consists in an impoverishment of the blood, especially the red cells, and a corresponding diminution in the specific gravity and in the amount of hæmoglobin. It is essentially a secondary anæmia, and occurs apart from disease of the blood-making organs. Infancy and childhood are themselves strong predisposing causes of anæmia, on account of the great demands made upon the blood in the rapid growth of the body.

**Etiology.**—The causes of anæmia embrace a wide range of pathological conditions. A child born of a delicate mother or of one suffering from tuberculosis or syphilis may show marked anæmia at birth. It may follow any severe hæmorrhage or occur in any of the blood dyscrasias, purpura, scurvy, etc.; also, the severe drain of prolonged suppuration, chronic nephritis, large serous effusions, many forms of diarrhœa and in malignant disease. Anæmia is often of toxic origin, sometimes being due to mineral poisons—lead, mercury, or potassium chlorate; more frequently it arises as the result of absorption of the products of excessive intestinal putrefaction. Certain of the specific infections, notably diphtheria, malaria, tuberculosis and rheumatism, produce a marked degree of anæmia, as one of their effects; also some of the intestinal parasites, particularly varieties of the tapeworm and hookworm.

Much more frequent in young children than any of the above are the anæmias due to improper feeding, rickets, and unhygienic surroundings. How important these causes are and how severe a grade of anæmia may be produced by them, is not usually appreciated. The physician is often led to suspect some serious organic or constitutional disease when none exists and to overlook such common conditions and obvious causes as those mentioned. Anæmia is seen when lactation is unduly prolonged. It is a frequent result of the long-continued use of milk or infant foods as the sole diet, given, as these often are, throughout the second or third year, for the reason that the child will take no solid food, because he is allowed to have the bottle. Lack of fresh air, confinement to overheated rooms and the crowding of young children in hospitals and institutions are common and important causes of anæmia.

**Symptoms.**—Anæmic children usually exhibit many symptoms of malnutrition. Their tissues are flabby; they are generally below average weight and suffer from digestive disturbances and chronic constipation. The associated nervous symptoms are many: headaches, indefinite pains, insomnia or disturbed sleep, general irritability and a high degree of nervousness. There is easy fatigue, shortness of breath on exertion, and sometimes fainting attacks. The peripheral circulation is poor; the hands



and feet are often cold. The pulse may be slightly irregular. Anæmic murmurs are heard over the base of the heart or the large vessels, and may be so loud even in infancy as to be mistaken for organic disease. A venous hum is sometimes heard in the neck. Epistaxis is not uncommon. The urine is scanty and sometimes pale. There may be enuresis. Œdema is rare in older children, but in severe anæmias of infancy it is often marked. In a certain number of cases, even of moderate severity, the spleen is much enlarged. Pallor of the skin and mucous membranes is present in most cases, but is not an accurate guide as to the degree of anæmia. This can only be determined by an examination of the blood.

*The Blood.*—There is a reduction of the number of red cells and to a still greater degree in the hæmoglobin. In a case of moderate severity the red cells are from 4,000,000 to 4,500,000, and the hæmoglobin from fifty to sixty per cent. In severe cases the red cells may fall to 2,000,000 or 2,500,000 or even lower, and the hæmoglobin to twenty or thirty per cent. These figures are not uncommon. The lowest I have seen is a reduction of the hæmoglobin to fifteen per cent and of the red cells to 1,400,000. The red cells are pale. There is usually poikilocytosis and anisocytosis; and, especially in infancy, a few normoblasts and megalocytes may be found (Plate XV, B).

There is generally a slight leucocytosis. The differential count of the white cells shows an increase in the lymphocytes, chiefly the small variety; the polymorphonuclear cells are relatively reduced in number.

**Prognosis.**—The course and termination of anæmia depend upon its cause. If this is one that can be removed, as in cases depending upon improper feeding and surroundings, very rapid improvement often takes place and prompt recovery. In the most severe cases death may occur, rarely from the anæmia, usually from some complicating disease.

In making a prognosis in a given case the general symptoms and the cause of the anæmia are much more important than the examination of the blood. If the digestive organs are in good condition and good surroundings can be secured, often, though the hæmoglobin and red cells are very greatly reduced, the prognosis is good. But in unfavourable surroundings and with a greatly disordered digestion, the outlook is much more serious.

Typical blood examinations of a moderate and of a severe case of secondary anæmia in a young child are as follows:

| SEVERE ANÆMIA.    |              | MODERATE ANÆMIA.  |              |
|-------------------|--------------|-------------------|--------------|
| Hæmoglobin        | 20 per cent. | Hæmoglobin        | 50 per cent. |
| Red blood cells   | 2,500,000    | Red blood cells   | 4,000,000    |
| White cells       | 12,000       | White cells       | 10,000       |
| Polymorphonuclear | 30 per cent. | Polymorphonuclear | 40 per cent. |
| Small mononuclear | 45 per cent. | Small mononuclear | 25 per cent. |
| Large mononuclear | 25 per cent. | Large mononuclear | 20 per cent. |
| Other forms       | 5 per cent.  | Other forms       | 5 per cent.  |

The treatment of all the forms of anæmia will be considered together at the close of the chapter.

#### CHLOROSIS.

Chlorosis is a primary or essential anæmia which usually occurs in young girls about the time of puberty. It is characterised by a peculiar greenish-yellow tint of the skin, and is not accompanied by emaciation. The changes in the blood consist in a very great reduction in the hæmoglobin without a corresponding diminution in the red corpuscles.

**Etiology.**—The exact cause of chlorosis is not yet understood. The disease rarely occurs in males; it is usually seen in girls between the fourteenth and seventeenth years, and more often in blondes than in brunettes. Heredity appears to be a factor in some cases. Other causes are occupations deleterious to health, such as employment in factories or confinement in ill-ventilated rooms; insufficient food or clothing; psychical disturbances, like grief, care, or fright; excessive mental or physical strain, and disorders of menstruation—although the latter are perhaps more frequently a result than a cause of the disease. Virchow first called attention to the fact that chlorosis might depend upon a congenital narrowing of the aorta, sometimes associated with a small heart. It is difficult to reconcile this etiology with the rapid recovery under appropriate treatment which is seen in most of the cases. Andrew Clark has advanced the view that the chief cause of chlorosis is constipation and the resulting absorption of toxic materials from the intestine.

**Lesions.**—Chlorosis is rarely fatal. In the few fatal cases the lesions noted have been dilatation of the right heart with hypertrophy of the left ventricle, a small aorta, small uterus and ovaries, and occasionally round ulcer of the stomach. Under the microscope there may be found a very marked degree of fatty degeneration of the heart muscle, and sometimes of the inner coat of the blood-vessels.

**Symptoms.**—The general symptoms of chlorosis are very much like those of simple anæmia. There are observed shortness of breath upon exercise, palpitation, syncope, attacks of vertigo, disturbances of digestion, amenorrhœa, and almost invariably constipation. The appetite is capricious, it being a peculiarity of these patients to crave all sorts of indigestible articles. Instead of the usual pallor of anæmia, the skin has a yellowish-green tint, from which the term "green-sickness" has arisen. Occasionally patches of pigmentation are seen. Anæmic cardiac murmurs may be heard in various situations, most frequently a systolic murmur at the base of the heart, and usually loudest over the pulmonic area. There may be a venous hum in the neck. In some marked cases there is evidence of slight cardiac dilatation, especially of the right heart, and there may be hypertrophy of the left ventricle. The pulse is weak and soft, œdema of the feet is frequent, and sometimes there is

slight albuminuria. In some cases there is fever. Nervous disturbances, such as vague, indefinite pains, attacks of migraine, supra-orbital neuralgia, various hysterical manifestations, and chorea, are common. Ulcer of the stomach is sometimes seen as a complication.

*The Blood.*—The specific gravity is reduced in proportion to the loss of hæmoglobin. The characteristic feature of chlorosis is a loss of hæmoglobin which is out of proportion to the reduction in the red cells. The hæmoglobin in an ordinary case is frequently as low as thirty-five or forty per cent, while the red cells may be 3,500,000 to 4,000,000, or even higher.

Morphologically the cells are pale with a wide central clear area. Poikilocytosis may be present, but is not marked; rarely normoblasts may be found. The presence of megalocytes is disputed. The leucocytes are usually unchanged in number and proportion, but the lymphocytes may be relatively increased.

**Prognosis.**—The course of the disease is essentially a chronic one, often lasting for a year. Relapses are quite frequent. Except when dependent upon congenital malformations of the heart and blood-vessels, these cases regularly recover when proper treatment can be carried out. A small number prove fatal by the development of tuberculosis or the occurrence of gastric ulcer.

**Diagnosis.**—A probable diagnosis is in most cases easily made from the etiology, the functional derangement of the heart, the colour of the skin, and a positive diagnosis always by an examination of the blood.

#### PSEUDO-LEUKÆMIC ANÆMIA OF INFANCY.

This form of anæmia was first described by Von Jaksch in 1889, and is by him believed to be peculiar to infants and young children. It is characterised by marked leucocytosis, marked reduction in the number of red cells and in the hæmoglobin, a great enlargement of the spleen, and sometimes a moderate enlargement of the liver and the lymphatic glands. This disease is not to be confounded with the pseudo-leukæmia of adults, or with Hodgkin's disease, which is a disease of the lymphatic glands with secondary anæmia, but without any leucocytosis.

The existence of pseudo-leukæmic anæmia as a distinct disease is denied by most of the authorities on diseases of the blood. It is to be regarded as a symptom-complex. All the reported cases can be classed as severe secondary anæmia, pernicious anæmia, or leukæmia.

**Etiology.**—Of the cases thus far recorded the majority have been between the ages of seven and twelve months. Of twenty cases collected by Monti and Berggrün, sixteen showed evidences of rickets and one was syphilitic. The exact cause of the disease is still unknown, and its essential nature is a matter of some doubt. Monti believed that it

might develop from the more severe cases of anæmia which are accompanied by leucocytosis, as he observed this condition before the development of pseudo-leukæmia and during its subsidence.

**Lesions.**—The most characteristic change is found in the spleen, which is very much enlarged, often forming an abdominal tumour of considerable size. It is firm, hard, and there may be evidences of perisplenitis. The microscope shows a simple hyperplasia. Enlargement of the liver is less constant, it being normal in more than half the cases. There is no relation between the size of the spleen and that of the liver. The hepatic cells are unchanged. Enlargement of the lymph glands has been noted in about half the reported cases, the swelling affecting the cervical, axillary, or inguinal glands; but it is rarely great. Inconstant changes in the bone-marrow have been described.

**Symptoms.**—*The Blood.*—The number of reported cases is as yet too small to make positive statements possible upon all points. The main features noted thus far are the following (Plate XV, C):

The specific gravity is lowered, the usual range being between 1.035 and 1.044. The reduction of the hæmoglobin is very great; in many of the cases it has been as low as thirty per cent, and in a few below twenty-five per cent.

The red cells are always diminished; in six of twenty cases they were below 1,600,000 (Monti and Berggrün). There is also great inequality in their size and shape. Nucleated red cells are found in considerable numbers; as a rule, these are chiefly normoblasts, but when the anæmia becomes more severe, it is usually the megaloblasts that predominate. The leucocytes vary from 20,000 to 50,000. They may show an increase in the mononuclear or in the polynuclear forms. The eosinophiles are usually increased, but not to the extent to suggest leukæmia. All varieties of cell degeneration are found.

The general symptoms of the disease develop slowly and with the usual signs of anæmia. In some cases the infants continue to be plump and well nourished. Pallor is usually very marked. Enlargement of the spleen is so great that it can hardly be overlooked if the abdomen is examined. The glandular enlargements are not marked, and in many cases are wanting altogether.

The course of the disease is essentially chronic. Cases have been seen in which pseudo-leukæmia developed from an ordinary severe simple anæmia in the course of a few weeks. The symptoms and blood changes generally come on slowly in the course of weeks or months, and sometimes remain nearly stationary for as long a period as several months, and then slowly improve. In other cases they grow gradually worse. In the cases going on to recovery, there is noticed improvement in the general symptoms coincident with a diminution in the size of the spleen, a reduction in the number of leucocytes, an increase in the red cells, the

hæmoglobin, and the specific gravity, and a gradual disappearance of the nucleated red cells.

**Prognosis.**—In Monti's list of twenty cases four proved fatal; one recovered, in which the proportion of leucocytes to the red cells had been one to twelve. The prognosis should be guarded, for, although improvement may take place, many patients die from intercurrent disease.

#### PERNICIOUS ANÆMIA.

This is the most severe form of anæmia known. Its cause and essential nature are as yet very imperfectly understood. It is characterised by quite uniform blood changes and by the general symptoms of a very marked anæmia, and it tends to go on from bad to worse, terminating fatally in the great proportion of cases.

**Etiology.**—Pernicious anæmia is a rare disease in childhood, and especially rare in infancy. In the cases which have been observed in early life the following etiological factors have been noted: It has been associated with hereditary syphilis and with severe rickets, especially when accompanied by a marked enlargement of the spleen. It has followed other diseases, especially grave disturbances of nutrition. Sometimes simple anæmia, when severe and of long standing, has gradually developed into the pernicious type. In a few instances parasites, particularly tapeworms, have been the cause. Pernicious anæmia has in some instances occurred in patients when no cause whatever could be assigned.

Many theories have been advanced in explanation of pernicious anæmia. The one which at present appears to have most in its favour is that the disease consists in a great destruction of the red blood-cells, particularly in the liver, and that this is brought about through the agency of some poison or poisons taken up from the intestine by the portal circulation. This has been advanced by Hunter and others in explanation of the peculiar deposit of iron found in the hepatic cells.

**Lesions.**—There is found a very high grade of anæmia in all the internal organs, fatty degeneration of the heart and blood-vessels, and sometimes also of the liver and kidneys, with numerous capillary hæmorrhages in the various organs. The most characteristic post-mortem change, however, consists in the deposit of iron in the hepatic cells. Its distribution is peculiar and unlike that seen in any other disease. The bone marrow is also markedly altered.

**Symptoms.**—*The Blood.*—The specific gravity of the blood in pernicious anæmia is constantly and considerably reduced, and its coagulability is feeble. The hæmoglobin is always reduced, usually it is as low as from twenty to thirty per cent. The red cells are always much diminished in number and generally to a degree greater than the reduction in the hæmoglobin. Their number is seldom greater than 2,000,000, and

frequently less than 1,000,000. Megalocytes are present, often in great numbers, and a preponderance of them is regarded essential to the diagnosis. Microcytes are rare. It is characteristic of pernicious anæmia that owing to the relatively high hæmoglobin content the red cells stain well, usually deeper than in normal blood. A striking feature of these cases is the presence of extreme poikilocytosis. Nucleated red cells are also present, megaloblasts in greater numbers than normoblasts. The red cells do not collect to form rouleaux.

The total number of leucocytes is markedly diminished, but the lymphocytes may be relatively increased. An occasional myelocyte may be found.

The general symptoms are those of a most intense anæmia. There is marked pallor of the skin and mucous membranes, with great weakness and prostration. Various anæmic heart murmurs are heard. There is dyspnœa, and usually the urine is scanty and of low specific gravity. There may or may not be emaciation. The late symptoms are hæmorrhages from the nose and other mucous membranes, subcutaneous ecchymoses with dropsy of the feet and ankles, and sometimes of the large serous cavities of the body, but without albuminuria. In many cases fever is present. This may be so high as to lead to the suspicion of some acute infectious process.

The course of the disease is, as a rule, more rapid than in adults, the duration being in most cases but a few months; it is marked by periods of exacerbation and remission. During the exacerbations all the symptoms are intensified, and as a rule some fever is present. During the remissions marked improvement may take place in all the symptoms and an increase in the hæmoglobin and red cells occur. In general, the progress of the disease is downward and sometimes the rate is very rapid. The only exceptions are the cases in which the disease depends upon some intestinal parasite, when improvement and even recovery may occur.

**Treatment of the Different Forms of Anæmia.**—In *secondary anæmia* the thing of the first importance is to discover and treat the primary condition upon which the anæmia depends. In infancy, special attention should be given to diet and hygiene, particularly with reference to an abundant supply of fresh air. The whole manner of life of these patients must be carefully studied and managed according to the directions laid down in the chapter upon Malnutrition, with which condition, especially in infancy, a very large number of these cases are associated. The general treatment referred to is often more important than the administration of the preparations of iron, which, however, should never be omitted.

The preparations of iron available for infants are the albuminate, bitter wine, sweet wine, saccharated carbonate, malate, and citrate. The

dose should be regulated according to the age of the child. Older children may take the same preparations as adults, especially reduced iron and Blaud's pills. Much benefit is seen from combining arsenic with iron, or from alternating the two. In addition to these remedies, cod-liver oil should be given if the condition of the digestive organs will permit.

In *chlorosis* more decided results are seen from the use of iron than in any other form of anæmia. Blaud's pills are here the favourite method of administration, and are advantageously combined with small doses of nux vomica and aloin to overcome the tendency to constipation. Arsenic is useful in these cases also. Great benefit in chlorosis results from change of air and change of scene, thus removing the patient from all sources of nervous excitement or disturbance. The general condition, diet, and habits of life should also receive careful attention, particularly the condition of the bowels.

Oxygen is a valuable adjuvant in the treatment of all anæmias not yielding to iron alone. It is important that the administration of iron should be continued for several months after the disappearance of all symptoms, on account of the tendency to relapse.

In the *pseudo-leukæmic anæmia* of infants, arsenic is decidedly the most valuable drug, but should be given in combination with iron. Fowler's solution is the best preparation for infants; the dose should rarely be more than one drop, which should be repeated four or five times daily after feeding, and continued for a long time. The general treatment of these patients is the same as in cases of simple anæmia. When rickets is present cod-liver oil and phosphorus should be added.

In *pernicious anæmia*, arsenic offers a much better prospect of improvement than does iron. Beginning with small doses, the amount should be gradually increased up to the point of tolerance, very much as in cases of chorea.

In every case of anæmia the most careful attention should be given to the general condition, particularly guarding against exposure to cold and dampness. The feeble circulation of these patients renders them peculiarly susceptible. Caution should also be given against much muscular exercise.

In many cases of anæmia of a severe grade, whether primary or secondary, transfusion offers a brilliant prospect of improvement and even recovery when no other treatment is of any avail.

#### LEUKÆMIA.

This is a disease in which the essential feature is a great increase in the number of leucocytes, with a moderate reduction in the number of red corpuscles, and the presence in the blood of cellular forms not found in health.

**Etiology.**—Leukæmia is a rare disease in childhood, but it has been seen even in early infancy. Its greater frequency in males holds good even in childhood. In a small number of cases heredity seems of some importance as an etiological factor. Leukæmia may follow syphilis, rickets, malaria, or even simple anæmia, or it may occur as a primary disease in children previously healthy. The cause is unknown.

**Lesions.**—The essential lesions of leukæmia are found in the spleen, the lymphatic glands, and the bone-marrow. In rare cases the most important changes are in the lymphatic glands, giving rise to the *lymphatic* form of leukæmia. In such cases the changes in the spleen or marrow may be slight or absent. Changes in the spleen and marrow are, however, usually associated, giving rise to what is known as the *spleno-myelogenous* form of the disease, which is the most frequent variety. The spleen is usually enormously enlarged, sometimes filling half the abdominal cavity. In the early stage it is soft, vascular, and of a dark-red colour; in the late stages it is firm and hard, and usually deeply fissured at its margin. There may be perisplenitis. On section, light-gray patches of lymphoid tissue may be seen scattered throughout the organ, and in some instances there may be wedge-shaped infarctions. The microscope shows thickening of the trabeculæ and deposits of lymphoid tissue, especially about the arteries. In the lymphatic form any of the external glands of the body may be affected, the cervical, axillary, and the inguinal, or the mesenteric, tracheo-bronchial, the tonsils, and even the lymph nodules of the tongue, pharynx, and intestines. The changes in the glands are generally those of a simple hyperplasia. The liver is enlarged in very many of the cases, chiefly from an infiltration with lymphoid tissue, which may be diffuse or may occur in patches. Less frequently similar lymphoid masses are seen in other organs.

**Symptoms.**—*The Blood* (Plate XV, D).—The colour is lighter than normal and its coagulability usually diminished. Generally the red cells are much reduced in number, although not to the extent seen in pernicious anæmia. The most important feature is the great increase in the number of leucocytes, which vary in form according as the type is spleno-myelogenous or lymphatic. The red cells are usually of normal size and a moderate number of normoblasts is found; the hæmoglobin is diminished.

In the spleno-myelogenous form the white cells may be from 100,000 to 500,000, but, especially under the influence of arsenic, a marked temporary diminution may occur, so that their number may be scarcely above the normal; both Ehrlich's and Cornil's myelocytes are present, and the presence of a large number of these is pathognomonic. The number of polymorphonuclear neutrophiles is greatly increased, although their proportion is diminished. The eosinophiles are very much increased in number, mononuclear forms being present. The number of lymphocytes is in-



creased, but they vary according to the type and stage of the disease; this is true also of the large mononuclear leucocytes. Mast cells are much increased in number, this being the most reliable diagnostic sign.

In the lymphatic form the lymphocytes alone are increased, so that the other white cells are relatively diminished. The increase is usually in the small lymphocytes, which form from eighty to ninety per cent of the leucocytes present. Myelocytes and mast cells are either present in small numbers or absent altogether.

The other symptoms of leukæmia in children resemble those in adults, with the difference that, as a rule, the progress of the disease is much more rapid in early life. In most of the cases the early symptoms are latent. A sudden and alarming hæmorrhage is sometimes the first thing to call attention to the serious condition. In other cases there are only the symptoms of general weakness and anæmia. Sometimes the splenic tumour or the enlargement of the lymphatic glands is first noticed. In the early part of the disease, the usual symptoms of anæmia are present—digestive disturbances, shortness of breath, weak and rapid pulse. Hæmorrhages may occur as an early or late symptom; they are most frequently from the nose, but severe hæmorrhages may occur from the stomach, the mouth, the intestines, or there may be ecchymoses upon the skin. The enlargement of the spleen may be sufficiently marked to form an abdominal tumour, so as to attract the attention even of the parents. The swelling of the liver is not so great. The lymphatic glands are enlarged only to a moderate degree, and in many cases this symptom is absent altogether. They are painless, movable, and usually several groups are affected.

The late symptoms are dropsy of the feet or general anasarca, hæmorrhages, diarrhœa, headaches, general weakness, and attacks of fainting. Fever is quite constant in the late stages of the disease, and the temperature may be from 101° to 103° F. The urine may contain albumin and casts. Vision is sometimes disturbed by the formation of leukæmic plaques in the retina. It is rare that there are any symptoms referable to the bones, although expansion and tenderness of the flat bones have been observed.

**Course and Prognosis.**—The course of leukæmia is chronic, and in most cases slowly progressive, but not always so. The prognosis is very bad, the great proportion of the cases in children proving fatal within a year from the first symptoms, in infancy sometimes in two or three months. There has been described by Epstein and others an acute form of the disease, proving fatal in a few weeks. The usual causes of death are exhaustion, hæmorrhages, and broncho-pneumonia.

**Diagnosis.**—This, in children, has to be made chiefly from simple anæmia with leucocytosis, and pseudo-leukæmic anæmia. Without a blood examination this is impossible. The chief reliance is to be placed

upon the enormous increase in the leucocytes, and especially upon the presence of numerous mast cells and myelocytes.

**Treatment.**—The general treatment of leukæmia should be the same as that of anæmia. Of the drugs now in use, arsenic has altogether the most testimony in its favour. It must be given in large doses and for a long period. Next to this in value come iron and cod-liver oil. Leukæmia, however, is in most instances very little influenced by treatment. The reported cures must be taken with some allowance, for most of these were published before the time when leukæmia was sharply differentiated from simple anæmia with leucocytosis and from the pseudo-leukæmic anæmia of infancy.

### HÆMOPHILIA.

Hæmophilia is an hereditary disease, in which there is a tendency to profuse or even uncontrollable bleeding from slight wounds, or sometimes even spontaneously. In many cases there is associated an inflammation of the joints. Persons so affected are known as "bleeders."

**Etiology.**—The hereditary tendency of the disease is very strongly marked, and it has often been traced through seven or eight generations. Males are much more frequently affected than females, the proportion being about twelve to one. In the matter of inheritance, the disease is most often transmitted through the mother, who, however, usually escapes herself. Patients suffering from hæmophilia may have nothing else about them that is abnormal. The exact nature of the disease is unknown. It has no connection with either purpura or scurvy. Although generally classed among the diseases of the blood, it has not been established that there are any constant changes either in the blood or in the blood-vessels. But there is probably either a deficiency of some element of the blood necessary to produce coagulation, or possibly an excess of some element interfering with coagulation.

**Symptoms.**—The first manifestations of hæmophilia are not often seen before the second year. The hæmorrhages of the newly born have no relation to this condition. The discovery of the disease is generally quite accidental. The first hæmorrhage may be traumatic or spontaneous. In traumatic hæmorrhages there may be very severe bleeding after so slight a wound as the drawing of a tooth; sometimes a large hæmatoma forms between the muscles as the result of a moderate contusion.

The following is the relative frequency of spontaneous hæmorrhages in 331 cases collected by Grandidier: Bleeding from the nose in 169, mouth in 43, intestines in 36, stomach in 15, urethra in 16, lungs in 17. There may be hæmorrhage from the skin or from any mucous membrane of the body. The attacks of spontaneous hæmorrhage are often periodical, and may be accompanied by arthritic symptoms resembling rheumatism. The severity of the hæmorrhages varies much in the dif-

ferent cases. From a slight wound a patient may bleed until he is exsanguinated, and even until death occurs. Such a result from the first hæmorrhage, however, is rare. In some cases the disposition to bleed is outgrown in later life. Grandidier states that, of 152 boys, over one-half died before reaching the seventh year. It is striking that when the disease affects females there is no tendency to excessive bleeding at menstruation or parturition.

**Treatment.**—The indications at the time of bleeding are, to arrest the hæmorrhage by the use of the ordinary surgical means—especially compression. Calcium lactate and gelatine may be used as described in the hæmorrhages of the newly born; but little benefit is to be expected from drugs. In extreme cases transfusion may be practised. Its effects are sometimes very striking. In convalescence after attacks of hæmorrhage, iron and general tonics should be given. In all patients who are bleeders everything which might by any means excite hæmorrhage should be avoided. The marriage of girls who inherit the disease should be discouraged.

#### PURPURA.

The term purpura is used to designate a condition in which there is a tendency to spontaneous hæmorrhages beneath the skin, from the various mucous membranes, and in some cases into the internal organs. The term *purpura simplex* is applied to those cases in which the hæmorrhages are limited to the skin; *purpura hæmorrhagica* to those in which there is in addition bleeding from the mucous membranes or visceral hæmorrhages. It is impossible to draw a line sharply between these two classes of cases, as the chief difference between them seems to be one of degree. Purpura is sometimes known as *morbus maculosus* or as *Werlhof's disease*.

**Symptomatic Purpura.**—This occurs in quite a variety of conditions, the hæmorrhages generally being limited to the skin, but not always so. These cases may be grouped in the following classes:

1. *Infectious.*—This form of purpura is very constantly seen in malignant endocarditis, in the hæmorrhagic forms of the various eruptive fevers—measles, scarlet fever, variola, vaccinia, and typhus—also in epidemic meningitis and occasionally in diphtheria, pyæmia, and septicæmia. The occurrence of hæmorrhages in these cases appears to depend upon an altered condition of the blood, which is a direct result of the infection, and it is a bad prognostic sign.

2. *Cachectic.*—Purpura occurs late in the course of many protracted and exhausting diseases, especially in infancy. It is most frequently met with in broncho-pneumonia, empyema, tuberculosis, ileo-colitis, in both the tuberculous and the simple forms of meningitis, and in malignant disease. It also occurs from apparently similar causes in several

of the diseases of the blood, particularly in leukæmia and pernicious anæmia. In most cases of cachectic purpura the hæmorrhagic spots are small, not very abundant, and occur either upon the abdomen or the lower extremities. This form is quite common in hospital practice, and is almost invariably indicative of a fatal result. In cachectic purpura the hæmorrhages are usually limited to the skin. The condition is undoubtedly dependent upon a deterioration in the blood, possibly also upon the condition of the minute blood-vessels.

3. *Toxic*.—Certain drugs, such as phosphorus, quinine, potassium chlorate, and sometimes others, may in rare cases produce hæmorrhages when long continued or in large doses. The hæmorrhage of jaundice may also be considered in this group.

4. *Mechanical* hæmorrhages, such as those occurring in pertussis or epilepsy, are sometimes classed with purpura. In convalescence from protracted illness there are sometimes seen, when patients first stand or walk, purpuric spots on the lower extremities. They may occur after the confinement of a limb in bandages or splints. In both these cases the cause is partly mechanical and partly due to the weakened condition of the blood-vessels.

5. *Neurotic*.—These cases are occasionally seen in diseases of the spinal cord and sometimes in hysteria in young adults, but very rarely in children.

**Primary Purpura.**—This occurs in children of all ages, being not uncommon in infancy. Hæmorrhages of the newly born have not generally been included in this class, although there are some reasons why they might well be. The age at which primary purpura is most frequently seen is from two to ten years. The sexes are about equally affected; of Steffen's 56 cases, 27 were males and 29 females. The disease may occur in children who are cachectic, rachitic, or anæmic, and in those whose surroundings are poor, but it has not, like scurvy, any close relation to diet. It may follow any acute disease, being associated most frequently with derangements of the stomach and bowels. Quite often the disease develops abruptly, without any assignable cause, in children previously healthy.

*Lesions.*—The external hæmorrhages may occur upon any part of the body. There are smaller or larger ecchymoses or an infiltration of the tissues with blood, which undergoes gradual absorption with the usual changes. With the hæmorrhages, various forms of inflammation of the skin may be associated, especially erythema and urticaria, with sometimes more or less œdema. Hæmorrhages from the mucous membranes are more frequent, because of the feeble resistance of the tissues. There are seen ecchymoses upon the visible mucous membranes which resemble those upon the skin. At autopsy they are occasionally seen in the trachea or bronchi, but more often in the digestive tract. In

The discharge of blood from the stomach or intestine may be accompanied by very severe attacks of colic and tenesmus. In some of these cases there are pains and slight swelling of the joints. Renal symptoms are generally present. The attacks of abdominal pain with purpura and the discharge of blood may come on paroxysmally every few days for a period of several weeks. They have been ascribed to thrombosis of the intestinal vessels. This is sometimes known as "Henoch's purpura."

Constitutional symptoms are present in most of the severe cases. There is usually fever, from 101° to 103° F., and sufficient prostration to keep the patient in bed. If the amount of blood lost is large, there are the usual symptoms of severe anæmia. The loss of blood may be sufficient to cause death, particularly in infants. Cerebral symptoms may depend upon anæmia or upon meningeal hæmorrhage. They are not frequent in this form of the disease. Edema, especially of the face and feet, may exist without albuminuria, and albuminuria may be present in cases in which there is no renal hæmorrhage.

In some of the cases beginning with severe general symptoms, and occasionally when the onset is mild, the patients after a few days pass into a typhoid condition with low delirium, great prostration, weak and irregular pulse, dry, cracked tongue, and high temperature. Such cases are almost always fatal. They are not to be confounded with ordinary typhoid fever complicated by purpura.

The course varies much in the different cases. It lasts from one to six weeks, the symptoms slowly subsiding, but often showing a strong tendency to recurrence. The prognosis depends upon the age of the patient, the extent of the hæmorrhage, and the presence or absence of septic symptoms.

3. The Hyper-acute Form (*purpura fulminans*).—This is a rare form, especially in young children. Its development is usually sudden, with a chill, vomiting, marked prostration, and high temperature. The purpuric spots come out with great rapidity, and in the course of a few hours or a day they may be very extensive. In addition to the ordinary subcutaneous hæmorrhages, bloody vesicles may form upon the skin. In many cases the hæmorrhages are limited to the skin, the mucous membranes and the viscera escaping altogether. There is no tendency to gangrene. Cerebral symptoms are invariably present and usually prominent; there may be delirium, dulness, stupor, and finally coma. The spleen is apt to be enlarged. The urine is nearly always albuminous. This form of purpura has all the characteristics of a general infectious disease, and it is almost invariably fatal.

4. The Gangrenous Form.—Sloughing is not common in purpura, but it is most often seen in the mucous membranes. Osler refers to two cases affecting the uvula. I once saw a slough which caused perforation of the soft palate. Wickham Legg reports a case with gangrene of the

prepuce. Gangrene of the skin is even less frequent, although cases have been reported even in young children. Charron's case was only three years old, and several others in children are collected in Gimard's monograph upon this subject. The gangrene may involve the skin only, or the subcutaneous tissues, and even the muscles. It has been seen upon the upper and lower extremities, and even upon the face, and may extend over quite a large surface. In some of the milder forms of purpura, gangrene results from some slight injury, such as a blow, the pressure from a bandage, or, in the nose, from the pressure of a tampon. These cases are almost invariably fatal. Those in which the sloughing is confined to small areas of the mucous membrane of the mouth often recover.

5. The Rheumatic Form.—The term "rheumatic purpura" (*peliosis rheumatica*) is applied to cases, not so common in children as in older patients, in which subcutaneous hæmorrhages, and sometimes bleeding from the mucous membranes, are associated with painful joint swellings. These are to be regarded as cases of rheumatism complicated by purpura. The joints most frequently affected are the knee and the ankle. The arthritic symptoms are usually less severe than in attacks of acute rheumatism. There may be present erythema exudativum or erythema nodosum or urticaria. Usually there are throat symptoms and fever, and frequently œdema of the face and eyelids with albuminuria. The spleen may be enlarged. The usual duration is from one to three weeks, and although relapses may occur, the cases usually recover.

Joint symptoms, particularly articular pains, are not infrequent in the course of milder attacks of purpura without the febrile symptoms mentioned. In severe cases extravasations of blood have been reported as occurring in the tissues about the joints, and even in the joints themselves, these being cases of true arthritic purpura. It is probable that, in the past, some cases of scurvy have been included in this group.

**Diagnosis.**—The rapid acute cases may be confounded with the hæmorrhagic forms of the various eruptive fevers. The ordinary subacute or passive forms are chiefly to be differentiated from scurvy. The diagnosis is not difficult, and the mistake need not be made if the essential features of scurvy are borne in mind—its dietetic cause, bleeding gums, hyperæsthesia, and deep rather than subcutaneous hæmorrhages which are usually near the joints.

**Prognosis.**—This depends very much upon the form of the disease. Of 128 cases of all varieties occurring in children in Steffen's collection, there were 40 deaths. In 12 cases of severe primary purpura reported by Gimard, there were 3 deaths and 9 recoveries. Purpura simplex is rarely fatal; cases of purpura hæmorrhagica usually recover unless marked febrile symptoms are present. The forms classed as typhoid,

gangrenous, and purpura fulminans are almost invariably fatal. The tendency to relapse exists in all varieties.

**Treatment.**—The treatment of symptomatic purpura should have reference to the cause of the disease. The mild cases of primary purpura usually recover promptly under a tonic plan of treatment. The more severe cases require confinement in bed, absolute quiet, and care to avoid exposure and even the slightest injury or extra pressure upon any part. Drugs do not seem greatly to influence the course of the disease. Those most frequently employed are supra-renal extract, hydrastis, hamamelis, aromatic sulphuric acid, the vegetable acids, ergot, and gallic acid. Whether or not it is true, as claimed by some, that all hæmorrhagic diseases are related to scurvy, the striking improvement seen in this disease from the use of fresh fruit and vegetables suggests their employment in purpura. In some cases very decided benefit seems to follow their use in the acute stage, but more particularly in convalescence. For hyperacute and gangrenous cases, little can be done except to treat the symptoms. Surgical means of arresting the hæmorrhage are rarely successful. Iron and arsenic should be used during convalescence.

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## CHAPTER II.

### *DISEASES OF THE LYMPH NODES (LYMPHATIC GLANDS).*

It is characteristic of infancy and childhood that the lymphoid tissues—tonsils, adenoids, external and internal lymph glands, and many smaller lymph nodules throughout the body—are prone to swelling and hyperplasia. While this tendency belongs to all children, in certain individuals it is so marked as to deserve a place as a distinct diathesis. It was formerly classed as one of the manifestations of “scrofula” or “struma”; but the proof that most of the manifestations once called “scrofulous” are really forms of local tuberculosis, makes it undesirable to use that term to designate the condition under discussion.

In robust children infectious processes of the nose, pharynx, or bronchi cause acute swelling of the lymph nodes in the neighbourhood, which rapidly subside when the cause is removed. In others, in whom this vulnerability of the lymphoid tissues exists, the hyperplasia in the lymph nodes is out of proportion to the exciting cause and continues after the cause has ceased to operate. Certain children have at birth an excessive development of lymphoid tissue, particularly in the region of the throat in the form of enlarged tonsils, adenoid vegetations of the pharynx, etc.

The influence of heredity in causing this condition is too often seen

to be passed over as a coincidence. Frequently the parents, during childhood, suffered from the same condition, and often every member of a large family of children is affected. They may be in other respects healthy, reared amid good surroundings, and show no evidence of any other constitutional disease. Any disease in the parents in consequence of which children are born with tissues having less than normal resistance, may be regarded in the light of a remote cause.

The condition is seen to perfection in children reared in institutions and in crowded tenements. It is more common in cities than in the country. Anything which produces malnutrition or lowers the general vitality of the tissues may be ranked as a cause. Rickets is often associated; sometimes it is to be reckoned as a cause, and sometimes both conditions depend upon the same causes.

During infancy, the lymphoid structures most frequently affected are those connected with the gastro-enteric and the bronchial mucous membranes; in later childhood it is those which are connected with the pharynx and tonsils.

The degree of enlargement of the lymph nodes which is sometimes found in the different situations has often led to a misinterpretation of them, particularly by those who only seldom see autopsies upon infants or young children. They have often been connected with pathological conditions or clinical symptoms with which they have really nothing to do.

Enlargement of the mesenteric glands and of the solitary follicles of the large and small intestine is very frequently seen in infants who have died from marasmus, and has been regarded as the cause of the wasting, while in reality it was only the consequence of the chronic intestinal indigestion which is an almost constant accompaniment of that condition.

As age advances we usually see retrograde changes in the different groups of glands unless they become the seat of tuberculous infection. Those connected with the digestive tract generally begin to diminish after the second year, and by the fifth or sixth year the enlargement has almost disappeared; while the tonsils, adenoid growths of the pharynx, and enlarged cervical glands are usually stationary after the seventh or eighth year, and undergo quite a marked atrophy about the time of puberty. The presence of these enlarged lymph nodes and the catarrhal condition of the mucous membranes with which they are associated, are important in relation to all acute infectious diseases which affect these mucous membranes. They bring about an increased susceptibility to scarlet fever, measles, diphtheria, diarrhoeal diseases, and most of all to tuberculosis.

In the following table are given the situation and drainage areas of the various groups of lymph nodes of the head and neck which play so important a rôle in infancy and childhood.



| DATE   | DESCRIPTION      | AMOUNT | BALANCE |
|--------|------------------|--------|---------|
| 1911   |                  |        |         |
| Jan 1  | Balance forward  |        | 100.00  |
| Jan 15 | Interest on loan | 5.00   | 105.00  |
| Jan 30 | Interest on loan | 5.00   | 110.00  |
| Feb 15 | Interest on loan | 5.00   | 115.00  |
| Feb 28 | Interest on loan | 5.00   | 120.00  |
| Mar 15 | Interest on loan | 5.00   | 125.00  |
| Mar 31 | Interest on loan | 5.00   | 130.00  |
| Apr 15 | Interest on loan | 5.00   | 135.00  |
| Apr 30 | Interest on loan | 5.00   | 140.00  |
| May 15 | Interest on loan | 5.00   | 145.00  |
| May 31 | Interest on loan | 5.00   | 150.00  |
| Jun 15 | Interest on loan | 5.00   | 155.00  |
| Jun 30 | Interest on loan | 5.00   | 160.00  |
| Jul 15 | Interest on loan | 5.00   | 165.00  |
| Jul 31 | Interest on loan | 5.00   | 170.00  |
| Aug 15 | Interest on loan | 5.00   | 175.00  |
| Aug 31 | Interest on loan | 5.00   | 180.00  |
| Sep 15 | Interest on loan | 5.00   | 185.00  |
| Sep 30 | Interest on loan | 5.00   | 190.00  |
| Oct 15 | Interest on loan | 5.00   | 195.00  |
| Oct 31 | Interest on loan | 5.00   | 200.00  |
| Nov 15 | Interest on loan | 5.00   | 205.00  |
| Nov 30 | Interest on loan | 5.00   | 210.00  |
| Dec 15 | Interest on loan | 5.00   | 215.00  |
| Dec 31 | Interest on loan | 5.00   | 220.00  |
| 1912   |                  |        |         |
| Jan 1  | Balance forward  |        | 220.00  |
| Jan 15 | Interest on loan | 5.00   | 225.00  |
| Jan 30 | Interest on loan | 5.00   | 230.00  |
| Feb 15 | Interest on loan | 5.00   | 235.00  |
| Feb 28 | Interest on loan | 5.00   | 240.00  |
| Mar 15 | Interest on loan | 5.00   | 245.00  |
| Mar 31 | Interest on loan | 5.00   | 250.00  |
| Apr 15 | Interest on loan | 5.00   | 255.00  |
| Apr 30 | Interest on loan | 5.00   | 260.00  |
| May 15 | Interest on loan | 5.00   | 265.00  |
| May 31 | Interest on loan | 5.00   | 270.00  |
| Jun 15 | Interest on loan | 5.00   | 275.00  |
| Jun 30 | Interest on loan | 5.00   | 280.00  |
| Jul 15 | Interest on loan | 5.00   | 285.00  |
| Jul 31 | Interest on loan | 5.00   | 290.00  |
| Aug 15 | Interest on loan | 5.00   | 295.00  |
| Aug 31 | Interest on loan | 5.00   | 300.00  |
| Sep 15 | Interest on loan | 5.00   | 305.00  |
| Sep 30 | Interest on loan | 5.00   | 310.00  |
| Oct 15 | Interest on loan | 5.00   | 315.00  |
| Oct 31 | Interest on loan | 5.00   | 320.00  |
| Nov 15 | Interest on loan | 5.00   | 325.00  |
| Nov 30 | Interest on loan | 5.00   | 330.00  |
| Dec 15 | Interest on loan | 5.00   | 335.00  |
| Dec 31 | Interest on loan | 5.00   | 340.00  |

ment of the thymus to a degree sufficient to be regarded as pathological, is not an infrequent condition. An association with rickets is often observed, but it is doubtful whether this is anything more than a coincidence.

Since the large thymus is so important a lesion, it is desirable to know what may be regarded as normal. The most extensive observations

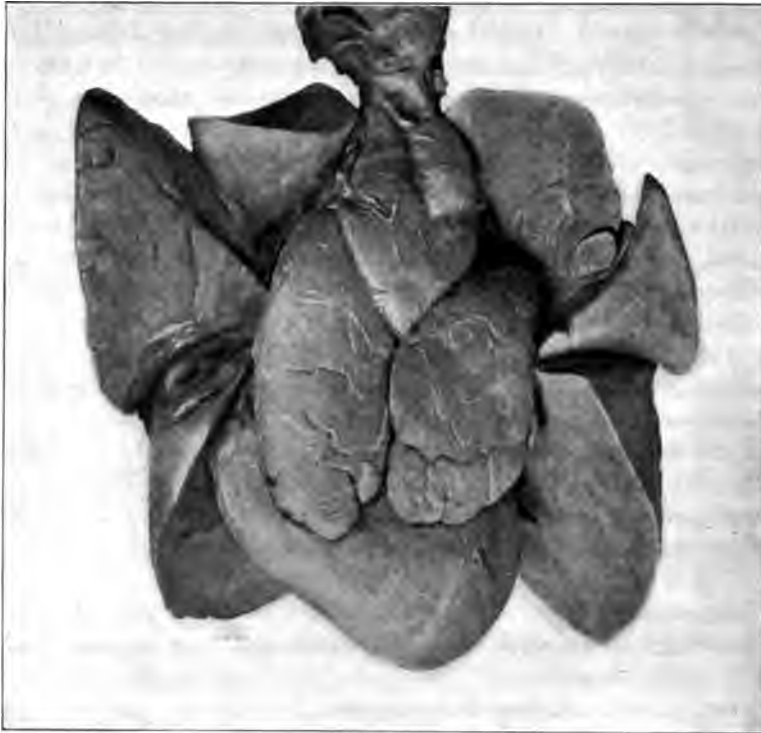


FIG. 160.—ENLARGED THYMUS. The lungs, heart, and thymus are shown in the picture. The lungs have been turned back, showing the two lateral lobes of the thymus overlapping the heart; the central lobe, above, covers the trachea. *History*.—Breast fed, male child, nine months old, well developed; ill less than twenty-four hours; dyspnoea, slight cyanosis, with death from asphyxia. T. 103° F. *Autopsy*.—Besides the large thymus there were present the general lesions of the status lymphaticus to a marked degree; lungs deeply congested.

upon this point have been made by Bovaird and Nicoll, who weighed the thymus in 495 consecutive autopsies in children under five years. They found that the weight was greatest at birth, the average being 7.7 grams. After this time the change in weight was very slight for the period of five years, the average for the entire 495 observations being 5.9 grams, which was about the same as the average for each of the years taken separately. Excluding cases in which the organ was so large as to be considered abnormal (10 grams or over), the average weight at birth was 6.5 grams; during infancy and early childhood, 4 grams. The re-

sults of these observations do not differ essentially from those of Friedleben, which have been so extensively misquoted. It may therefore be assumed that the average weight of the normal thymus at birth is from 6 to 7 grams; from birth to five years, from 3 to 4 grams. Anything over 10 grams may be considered abnormal.

In the status lymphaticus the thymus is often from five to ten times larger than normal. In the marked cases its weight is from 30 to 40 grams; in the less marked cases from 10 to 20 grams. The appearance of the enlarged thymus is well shown in the accompanying illustration (Fig. 160). A thymus of the size shown weighs about 45 grams, or  $1\frac{1}{2}$  ounces. In this instance it was nearly as large as one of the lobes of the lung. In general appearance, the enlarged thymus is rather more vascular than normal, but other than hyperplasia, shows no constant or essential changes, either by gross or microscopical examination.

The lymph nodes of the tracheo-bronchial region are greatly enlarged, often to the size of small cherries, and are found in great clusters. Those of the mesenteric region may be still larger. Peyer's patches are very prominent, and the solitary follicles of the small intestine appear like mustard seeds upon the folds of the mucous membrane. Those of the colon are also very prominent. The lymphoid tissues about the pharynx and all the lymph nodes of the body are greatly hypertrophied. The spleen is usually enlarged, with prominent follicles. There are no other constant changes. Those present are usually accidental, depending upon the cause of death.

**Symptoms.**—In very early infancy this is one of the explanations of sudden death occurring after slight causes, and in some cases without any apparent cause.

Death may be attributed to overlying, to asphyxia from food, or to some other condition affecting respiration, or infants are simply found dead in their cribs.

Even in those who live until they are several months, sometimes several years, old, there may be nothing in their condition to indicate the presence of the status lymphaticus until something acute occurs. This may be in the nature of a slight accident, a surgical operation of a trivial character, the administration of an anæsthetic, or some acute disease, frequently one affecting the respiratory tract. The symptoms associated with this condition are most frequently of a nervous character, usually attacks of convulsions, or they affect the respiration, causing paroxysms of dyspnœa, cyanosis, and even asphyxia. A frequent history is somewhat as follows: A child previously regarded as healthy, often well nourished and perhaps entirely breast fed, is taken with convulsions followed by high fever, preceding which there may have been some pulmonary symptoms suggesting a commencing broncho-pneumonia. The convulsions recur at short intervals; the temperature re-

mains steadily high; the signs in the lung are few and not proportionate to the other symptoms; and death occurs in from twelve to thirty-six hours often in convulsions.

In other cases convulsions are absent and the prominent symptom is asphyxia, which comes in paroxysms and may be so complete as to lead to the suspicion of laryngeal obstruction. If intubation or tracheotomy is performed, no relief follows. The child may die in the first severe attack, which may be preceded for a few hours by moderate dyspnœa, or may come on almost without warning. It is more frequent, however, for the first attack to be less severe, the child perhaps being resuscitated with some effort, after which he may breathe almost as well as usual. In a few hours the attack of asphyxia is repeated; after several of these, each one growing more severe, death occurs. In these cases the elevation of temperature is usually slight and may be wanting.

Symptoms similar to the above but of less severity and resulting in recovery would suggest status lymphaticus, although the diagnosis can not be established.

The cause of the symptoms is not definitely known. The asphyxia has been ascribed to pressure of the large thymus upon the lungs, the trachea, the pneumogastric nerves, or the auricles of the heart. Pressure would certainly seem to be one factor in the production of the dyspnœa. Further evidence in support of this is obtained by the relief afforded by an operation in which the anterior mediastinum is opened and the thymus raised and either fixed to the sternum or removed. This has been done in several instances with striking benefit.

In other cases, although the thymus may be quite as large as in those just described, the evidences of obstructive dyspnœa are much less and may scarcely be noticed.

There is another group of cases, perhaps the largest of all, in which there are no symptoms distinctly referable to the status lymphaticus, and yet this condition appears to be the factor which determines the fatal outcome of what was apparently an infection or an inflammation of only moderate severity. What is seen here is simply a greatly diminished resistance to disease. In these cases it is only the autopsy which reveals the explanation.

**Diagnosis.**—The diagnosis of the status lymphaticus is very uncertain. In some cases of marked enlargement it is possible to make out the enlarged thymus by percussion, but this is always difficult on account of its proximity to the lungs and trachea. We may suspect this condition during life; we can hardly do more. Marked enlargement of the tonsils and the adenoids exists so frequently without thymus enlargement, that this can hardly be regarded as suggesting the condition. The hyperplasia of the tracheo-bronchial or mesenteric lymph nodes or of the follicles of the intestine produces no especial symptoms.

**Prognosis.**—While this condition apparently may exist for an indefinite time without producing any symptoms, it undoubtedly often determines a fatal outcome of what might otherwise have been a mild illness or a trivial accident. It is especially important in connection with acute bronchitis and broncho-pneumonia, with attacks of convulsions, with the shock of slight operations, and with the administration of anæsthetics, particularly chloroform. It is one of the most frequent explanations of unexpected death from slight causes, such as an exploratory puncture or the injection of antitoxine.

At present no known treatment has any influence upon the condition.

#### SIMPLE ACUTE ADENITIS.

This is an acute inflammation of the lymph nodes which in infancy frequently terminates in suppuration. A certain amount of inflammation of the lymph nodes occurs in children in all acute processes affecting the mucous membranes, especially when they are severe or prolonged. Those in connection with the various internal organs are considered with the diseases of the organs. Acute inflammation of the external nodes is of sufficient frequency to require separate consideration. While this is probably always secondary to some pathological process in the skin or mucous membranes, the primary condition may be so slight as to be overlooked, and the adenitis may be the more important condition or may even assume the appearance of a primary disease. It is particularly in infants that this is seen, and it depends upon the unusually active absorption and upon the susceptibility of the lymphoid tissues at this age. The cervical glands are frequently affected, and occasionally those of the axillary and inguinal regions.

**Etiology.**—Acute adenitis occurs in children of all ages in connection with diphtheria, scarlet fever, measles, and influenza. In such cases it is often severe, and after scarlet fever, frequently terminates in suppuration. With the simple acute catarrhal processes of the pharynx and rhino-pharynx adenitis also occurs, but it is usually mild and rarely ends in suppuration. In infancy, on the other hand, acute adenitis from simple catarrh is not only very common but often severe, and frequently terminates in suppuration. Ulcerative stomatitis, carious teeth, eczema of the scalp or traumatism, may excite adenitis in children of all ages. Axillary adenitis may result from vaccination; inguinal adenitis, from vaginitis.

Of 109 cases of acute adenitis from my records, not including any associated with diphtheria, measles, or scarlet fever, more than three-fourths occurred in the first two years, and half of them in the first year of life. This susceptibility of infants is very striking. The disease occurs frequently in those who are in other respects perfectly healthy,

and often when the evidences of disease of the mucous membrane are slight. This is true not only of the cases of cervical adenitis, but also of others in which the inguinal glands are involved. The inflammation is excited in most of these cases by the absorption of pyogenic germs, usually staphylococci or streptococci, from the mucous membranes or skin.

**Lesions.**—The changes taking place in the glands are acute congestion, with swelling, œdema, and active hyperplasia of the lymphoid elements. The process may terminate in resolution or in suppuration according to the intensity of the infection and the susceptibility of the tissues. When severe enough to cause suppuration, the adenitis is accompanied by considerable inflammation of the surrounding cellular tissue.

In the series of 109 acute cases to which I have referred, not including the specific infectious diseases, 96 were cervical, 9 were inguinal, and 4 axillary; sixty-two per cent terminated in suppuration, the latter being nearly all in infancy. Suppurative otitis was present in sixteen per cent of the cases. Suppurative retro-pharyngeal adenitis (retro-pharyngeal abscess) was seen in several cases.

In infancy the disease is usually unilateral, or, if bilateral, the glands of one side are more severely affected than those of the other. Suppuration is nearly always of one side, and usually the abscess starts in a single gland.

**Symptoms.**—The symptoms and course of the adenitis of the specific infectious diseases belong to their clinical history. Suppuration is infrequent, except after scarlet fever. It is very rare after diphtheria.

The typical cases of acute adenitis are those which occur in infancy. There are present the symptoms of the original disease—usually catarrh of the nose or rhino-pharynx, mouth, or ear, which may not be very severe, and sometimes is overlooked. The glands most frequently affected are the deep cervical group. The tumour appears just below the angle of the jaw at the anterior border of the sterno-mastoid muscle (Fig. 161). The swelling during the acute catarrh is not rapid or great, but continues after the original process has subsided until it reaches the size of a walnut or even larger. In the most acute cases there is marked



FIG. 161.—ACUTE SUPPURATIVE ADENITIS IN AN INFANT ONE YEAR OLD. Showing the most frequent situation of the tumour in the cervical region.

inflammation of the periglandular cellular tissue, with pain, tenderness, and extra heat. If suppuration occurs, it is generally evident in the latter part of the second week, but sometimes it may be as late as the



FIG. 162. — ACUTE SUPPURATIVE ADENITIS (INGUINAL) IN AN INFANT THREE MONTHS OLD.

third or even the fourth week. In the axillary or inguinal region (Fig. 162) the symptoms of adenitis are essentially the same as in the neck. In the inguinal cases the degree of catarrh of the mucous membrane is often very slight.

Most cases run their course with slight fever and few general symptoms; but in young infants the constitutional symptoms are often severe and the physician may be in doubt whether the local process is sufficient to explain them. The temperature may be from 102° to 104° F. for several days, with considerable prostration, which is much increased if there is complicating otitis. After suppuration, if freely

opened at the proper time, the abscess heals rapidly and permanently, a sinus being rare. Occasionally infection extends from one gland to another, and a succession of these glandular abscesses occurs.

In the non-suppurative cases the swelling may be even greater than in those which suppurate; but it is less diffuse and apparently limited to the gland. It subsides slowly in the course of from four to eight weeks, often leaving a small tumour which may be apparent for several months. In susceptible children recurrent attacks of acute inflammation may lead to chronic enlargement which may last indefinitely. These glands do not become cheesy, except from subsequent tuberculous infection.

The acute cases in infancy in which suppuration occurs, appear to recover about as promptly and quite as completely as those terminating in resolution, although in the former the constitutional symptoms are more severe.

**Diagnosis.**—This is usually easy if it is remembered that, with the exception of the specific infectious diseases, and occasionally local causes like eczema of the scalp, carious teeth, etc., acute suppurative adenitis is essentially a disease of infancy. I have often seen it mistaken for mumps when the swelling was severe, but on close examination there is but little resemblance between the conditions. The disease is usually acute, and has little in common with the slow suppuration seen in later

childhood from the breaking down of tuberculous glands. In the occasional cases seen in which the disease runs a slow course a diagnosis from the tuberculous form may be aided by a tuberculin test.

**Treatment.**—Prophylaxis requires that in all acute catarrhs the mucous membrane should be kept as clean as possible by the use of nasal or pharyngeal sprays, or by syringing with simple solutions like Dobell's or Seiler's, or one of common salt.

In the stage of acute inflammation very hot applications or an ice-bag may be used for the relief of pain. It is very doubtful whether either of these means has much influence in preventing suppuration. If abscess forms, incision should be deferred until pointing has taken place. If this plan is followed, refilling is rare. A simple incision with proper aseptic treatment is all that is required. Curetting may be done if there is much broken-down tissue present, but it is not usually necessary. In most of the cases the abscess promptly heals and a perfect cure takes place. In cases which do not suppurate, absorption may be promoted by the internal use of the iodide of potassium in full doses—gr. x daily to an infant of one year. I confess rarely to have seen any benefit from painting with iodine or from inunctions of iodine ointment or the oleate of mercury. If adenitis is secondary to carious teeth, eczema, or ulcerative stomatitis, these conditions should receive appropriate treatment. Such cases do not usually suppurate, but subside rapidly when the primary cause is removed.

#### SIMPLE CHRONIC ADENITIS.

This consists in a simple hyperplasia of the lymph nodes. There are considered here only the external glands, but those of the cavities of the body are affected in a similar way, in diseases of the mucous membranes with which they are connected.

Simple chronic adenitis is not nearly so frequent as the acute form even in infants and young children, and it is rare after the fifth year. It may follow one or more attacks of acute adenitis, or it may result from subacute or chronic inflammations of the skin or of the various mucous membranes, infection from which causes the acute form. The most frequent subjects are children who have the diathesis described as "lymphatism."

**Symptoms.**—The glands upon both sides of the neck are usually involved, and more often a group than a single gland. The degree of swelling is not generally great, being much less than in acute adenitis, and usually less than in the tuberculous form. There are no constitutional symptoms. Hypertrophy of the tonsils and adenoid growths of the pharynx are frequently present. There is no tendency to suppuration or caseation. The swelling usually increases slowly for one or two months, then remains stationary for about the same length of time, after



which it slowly subsides. A subacute course is more frequent than a very chronic one.

**Diagnosis.**—These cases are especially to be distinguished from those of tuberculous adenitis. The most important points for differentiation are, that they occur most frequently in children under two years, a period when tuberculous adenitis is not common; some definite exciting cause is usually present; caseation and suppuration do not occur; the glands do not become adherent to the skin or to the deeper tissues; they enlarge much more rapidly than do the non-caseating tuberculous glands; and they are influenced to a much greater degree by constitutional treatment. The children do not respond to tuberculin tests.

**Treatment.**—Operative measures are not called for in simple adenitis; but there are some cases in which operation is to be considered if a thorough trial of other measures for two or three months has been without benefit. Local causes usually found in the pharynx, nose, or mouth should be removed if possible. Often more can be accomplished by removal to a climate in which the child's catarrhal symptoms are relieved than by all else. Little benefit is seen from local applications. The most useful internal remedies are, the syrup of the iodide of iron (twenty drops three times a day to a child of four years), and arsenic (two or three drops of Fowler's solution three times a day). Cod-liver oil should be given except during warm weather.

#### SYPHILITIC ADENITIS.

It is quite rare that a marked degree of glandular enlargement is seen as a symptom of hereditary syphilis; indeed, it is so rare that it is often forgotten that chronic multiple glandular enlargements are ever due to this disease. In the few examples that have come under my observation, this has been a late symptom of hereditary syphilis. The glandular enlargements were cervical and multiple, and the degree of swelling was often marked. They may be associated with disease of the bones or of the mucous membrane of the throat or of the nose, or without signs of such disease. The diagnosis of syphilis rests upon the association of other late manifestations of the disease—keratitis, periostitis, deformities of the teeth, the Wassermann reaction, and the prompt improvement under anti-syphilitic treatment. In their local appearance they resemble tuberculous glands.

#### TUBERCULOUS ADENITIS.

(*Scrofula.*)

Tuberculous disease of the lymph glands of the cavities of the body is discussed elsewhere; only that of the external glands is here considered. This condition presents some striking peculiarities: it is rela-

tively rare in infancy, although a frequent form of tuberculosis in older children; it often exists as the only apparent tuberculous lesion in the body. In the great majority of cases it is the cervical glands which are affected.

**Etiology.**—The age at which tuberculosis of the cervical lymph glands is usually seen is from three to ten years. In my experience with tuberculosis in infancy, the external glands are rarely involved, while the bronchial glands are almost invariably the seat of infection.

Local conditions favouring infection are adenoid growths of the pharynx, chronic pharyngitis, and hypertrophied tonsils; less frequently chronic otitis, chronic conjunctivitis, and pathological processes of the skin or the mouth, such as eczema of the face or scalp, ulcerative stomatitis, carious teeth, etc. That the pharynx is the most frequent seat of primary infection, is shown by the fact that the deep cervical glands are generally first affected. The question often arises whether the process is at first a simple one, and later becomes tuberculous, or whether it is tuberculous from the outset. My own belief is that in most cases the process is a tuberculous one from the beginning.

Children who are by inheritance predisposed to tuberculosis and those also who are prone to glandular enlargements—two conditions which are by no means identical—are the ones most liable to be affected. Attacks of acute infectious diseases, particularly measles, scarlet fever, and influenza, frequently play the rôle of exciting causes.

The age of those affected corresponds very closely with that at which children are most often seen with hypertrophied tonsils and adenoid growths of the pharynx. The subsidence of symptoms about the time of puberty, is also characteristic of both conditions. Of forty-five cases of tuberculous cervical adenitis in children studied by Park, twenty-five showed the human type of bacillus, and twenty the bovine type. This is in striking contrast with the results found by him in other forms of tuberculosis in children and points strongly to food infection as a cause.

**Lesions.**—It has been already stated that in the great majority of cases the cervical lymph nodes are involved, and generally they are the only ones affected. In 155 cases of tuberculous glands in the series reported by Treves, those of the neck were the seat of disease in 145 and the only seat in 131; those of the axilla were involved in 17, but alone only in 4; the groin in 8, and alone in 6. The nodes first affected are most frequently the upper set of the deep cervical group; sometimes, however, it is the superficial nodes of the submaxillary, or the parotid group, and occasionally the submental or the pre-auricular. The chain of deep cervical nodes which is involved, follows the carotid artery, and often extends some distance below the clavicle. These deep nodes are sometimes connected with the bronchial group.

The process in all tuberculous glands is essentially a chronic one, but pathologically the cases may be divided into two groups, corresponding somewhat to the forms of disease seen in the lungs. In one group the process is more rapid, and tends to early caseation and softening; the products of inflammation are mainly cellular, and the amount of fibrous tissue is small. In another group the course is slower, and fibrous tissue predominates, caseation and softening being infrequent.

In the first group the glands in the early stage are swollen, of a pale pink colour, and homogeneous; later they become more firm, and show, as the first gross evidence of tuberculous deposits, small grayish-white spots, which are generally numerous and scattered through the affected gland; these spots enlarge, and may coalesce to form one large gray mass, involving nearly the whole gland. Subsequently there is caseation and then softening, usually beginning in the centre of the caseous area. Inflammation within the gland is followed by that of the surrounding tissues, which may result in adhesions or in the formation of a periglandular abscess. The first change in the gland is the production of epithelioid and giant cells, about which there is a zone of small round cells; cheesy degeneration then begins in the centre. The caseous masses may become encapsulated by the production about them of fibrous tissue; or softening may occur at one or more foci, and an abscess form. Such an abscess contains curdy material but very little true pus, the contents being chiefly detritus from the broken-down node. Tubercle bacilli are usually more numerous in the early stages of the process, but are often difficult of detection in broken-down tissues, and the curdy pus is sometimes sterile. As the glands soften, the process gradually extends from the centre to the surface, and they become adherent to the surrounding structures—blood-vessels, nerves, or the fascia—they fuse together and form large knotty masses, and when they ultimately break down they lead to the formation of an abscess in the cellular tissue, finally involving the skin. In the form of suppuration which occurs in and about tuberculous nodes, an important part is often played by other bacteria, usually the staphylococcus or the streptococcus.

In the second group of cases, where the process goes forward more slowly, the changes are not quite the same, the essential difference being that the amount of fibrous tissue is much greater. These nodes are not so vascular; they are tough and hard, appearing like small fibrous tumours. The capsules are greatly thickened, and under the microscope is seen fibrous tissue arranged in concentric layers, often inclosing small caseous masses. These nodes less frequently form adhesions to the surrounding tissues, and consequently are freely movable, while suppuration is quite exceptional. Although the separate tumours are much smaller than in the first group, the glandular mass is often a large one, because of the number of glands involved.

It is seldom in either group of cases that the process is limited to a single node or even to two or three nodes. Very often an entire chain is involved (see Fig. 163).

Tuberculous infection of the lymph nodes may terminate in resolution, encapsulation, calcification, or suppuration. The inflammation may subside before caseation has taken place and the inflammatory products undergo absorption. After caseation has occurred the masses may become encapsulated and contract to small fibrous nodules. Calcification of the glands in this location is rare. In other cases caseation is followed by breaking down, liquefaction, and an external abscess. The course which the local disease takes will depend upon the intensity of the infection and the general vigour and resistance of the child. There is seen in most cases a tendency of the inflammation to subside spontaneously about the time of puberty. Cure has sometimes followed an acute attack of intercurrent disease, such as erysipelas of the face, and even scarlet fever.

**Symptoms.**—In the early part of the disease there are no symptoms but the glandular swelling, and this begins very gradually. In most cases both sides are involved, but as the disease progresses the advanced changes are usually confined to one side. The enlargement is seldom continuous; it often increases for a time and then remains stationary or even diminishes, to take a new start from the stimulus of some fresh infection of the mucous membrane with which the glands are associated, such as an attack of measles or influenza, or simply from a deterioration in the patient's general health. During exacerbations, the glands may be painful and tender, and show the usual signs of local inflammation.

The whole course of the disease varies from several months to as many years. Treves gives three and a half years as the average duration when suppuration occurs. The glands first affected are usually those situated near the bifurcation of the common carotid artery. Such tumours usually make their appearance just in front of the sterno-mas-



FIG. 163.—POSTERIOR CERVICAL CHAIN OF TUBERCULOUS LYMPH NODES. The upper one showed giant cells and extensive cheesy degeneration; one at the middle showed early tuberculous changes—cell infiltration, giant cells, and a small area of cheesy degeneration; the lowest node showed one small tubercle with a cheesy centre. Child two and a half years old. (Dowd.)

toid muscle—sometimes behind it—and at the level of the upper border of the larynx or the hyoid bone. In the more rapid cases the tumours usually attain a considerable size in three or four months, sometimes in half that time. The usual size reached is from that of an almond to an English walnut. At first the tumours are movable and preserve their distinct outline; later they become adherent, first to the deeper tissues and to each other, finally to the skin, and there is formed an irregular nodular mass in which it is sometimes difficult to make out the individual glands. As the process approaches the surface there are small spots of softening; then there is distinct fluctuation; the skin becomes discoloured and finally gives way, and there is a discharge of thick, curdy pus, which may continue for an indefinite time, until the whole of the broken-down gland has been thrown off. This course is repeated with each successive gland which breaks down. In cases progressing more slowly the glands become adherent chiefly to one another, and suppuration is less frequent.

In what proportion of tuberculous lymph nodes suppuration occurs, it is difficult to say. Like other tuberculous lesions in the body, this one is more frequent than was once supposed; and in the past most of those which did not break down were not classed as tuberculous. It is probable that of the cases allowed to run their course about one-half terminate in suppuration. Two forms of suppuration occur in connection with tuberculous glands—one an abscess of the gland proper, the other outside of and usually over it. In a typical case of the first variety, the gland is distinctly outlined and often superficial, there is very little inflammation, the spot of softening and fluctuation is small, and the pus discharged is always curdy. In the second variety the abscess is preceded by a more diffuse swelling, and the outline of the gland may not be made out; the signs of inflammation are more marked, the area of fluctuation is larger, and the pus is more like that of any ordinary abscess. Often the two varieties are combined; as when a gland beneath the deep fascia breaks down and there is formed directly over it an abscess in the cellular tissue, which communicates through a narrow opening with the gland beneath. In such cases the sinus continues open for a very long time, until the whole of the gland has been discharged. If healing occurs before this, the cicatrix soon breaks down.

Where abscesses are allowed to open spontaneously, large, irregular, and usually very intractable ulcers form. The skin is undermined for a considerable distance, and it has an unhealthy appearance. Such ulcers sometimes continue for many months in spite of all treatment, particularly if the patient's general health is poor. The scars left after them are large and unsightly, and sometimes positively deforming (Fig. 164). Their appearance is quite characteristic. They often have many tabs of skin attached to them; they may form prominent ridges which undergo

contraction like those after burns; they are of a purplish-red colour, and adherent to the deeper tissues. They are often sensitive and painful. As time passes they atrophy and become less conspicuous, though they remain throughout life.

The general health of children with tuberculous glands may be much or little affected, and not a few remain in good condition throughout the whole course of the disease, particularly when suppuration does not occur, but sometimes even when it is protracted.

**Prognosis.**—Tuberculosis of the external lymph nodes is seldom if ever the direct cause of death; although the course is often very protracted, ultimate recovery can usually be predicted. As previously stated, it is surprising

that this process is so frequently the only tuberculous lesion in the body. Treves states that the percentage of those who die from general tuberculosis is so small that this danger is not to be considered an argument for operation. Poore reports that of 58 cases treated by operation, only 2 were known to have died from tuberculosis. Dowd has collected reports of 309 cases treated by removal more or less complete, whose histories were followed for several years after operation. Of these, 202, or 65.4 per cent, were apparently cured; 57, or 18.4 per cent, were living, though suffering from either local or general tuberculosis; 50, or 16.2 per cent, died of tuberculosis. These statistics surely do not support the hopeful views of the writers first quoted, but they are, I think, more in accord with general experience.

**Diagnosis.**—The diagnostic features of tuberculous glands are the age of the patient—usually from three to ten years—the site of the primary swelling, the indolent course, the trifling original cause, and the disposition to slow caseation, softening, and abscess. The tuberculin reaction is of great assistance. The cases of simple hyperplasia are



FIG. 164.—CICATRICES FOLLOWING A NEGLECTED CASE OF TUBERCULOUS ADENITIS, IN A GIRL SEVEN YEARS OLD. There is also a tuberculous patch upon the skin of the cheek in a very frequent location.

usually in children under three years, their progress is much more rapid, there is often some definite cause, and in most cases they nearly or quite disappear in the course of three or four months. They suppurate, if at all, during the first month. Syphilitic disease is to be recognised mainly by discovering the evidence of syphilis elsewhere, and by the effect of treatment. In Hodgkin's disease, glandular groups in other parts of the body are involved simultaneously or in rapid succession. There are no signs of inflammation or caseation; and the swellings are accompanied by very marked and definite constitutional symptoms—*anæmia*, *emaciation*, and general prostration. Malignant growths are very rare; they increase rapidly, often attaining a great size in a few months.

**Treatment.**—The general treatment of tuberculous glands is to put the child under the very best surroundings possible. The seaside has a great reputation for such cases, and no doubt the majority do very well there; but some are benefited even more by a dry, mountain climate. At all events, a child from the city should be sent into the country whenever this is possible. Internally the only remedies which have any special virtues are cod-liver oil and the syrup of the iodide of iron. The latter should be given in full doses, i. e., twenty or thirty drops, three times a day, to a child of six years. Arsenic and iron are useful as general tonics. Local applications are of little value and most of them positively harmful; painting with iodine and poulticing should be discarded altogether. The parts should be rubbed or handled as little as possible.

It is important in every case to remove from the nose and throat all sources of local irritation. Hypertrophied tonsils and adenoid tissue of the pharynx should receive attention, also any pathological conditions in the nose, such as hypertrophy of the turbinated bodies, and chronic otitis, chronic conjunctivitis, carious teeth or ulcers in the mouth. All these, if they do no more, keep up a constant glandular irritation, and produce conditions which are most favourable for the activity of the tubercle bacillus.

*Operative Measures.*—These are indicated if, after two or three months of constitutional treatment, the glands affected continue to increase in size and number, or if softening occurs. The advantages of operation over leaving the case to Nature are, that it leaves a clean scar instead of a large, irregular one; that it shortens the disease and prevents the long, tedious suppuration of cases left to themselves; that it is a radical measure; and that it avoids the danger of general infection by removing the tuberculous focus.

The best results follow operation when it is done early before the skin is involved or the glands have softened or have formed extensive adhesions to the great vessels and neighbouring structures; also where a chain of glands is involved and where the inflammatory process is slow or indolent. If performed early a thorough operation by a good surgeon

in the majority of cases will result in a permanent cure. However, the operation is not contraindicated in cases which have gone on to a later stage, although the results may not be quite so satisfactory.

Glandular abscesses should in all cases be opened as soon as pus forms, to prevent the extensive undermining of the skin, which is so likely to occur. The opening should be a small one, and all squeezing of the gland or surrounding tissues avoided.

#### HODGKIN'S DISEASE.

*(Pseudo-Leukæmia.)*

This is a rare disease in which there is a general hyperplasia of the lymphatic glands throughout the body, with growths of lymphoid tissue in the spleen, liver, and other internal organs. It is accompanied by marked anæmia, is progressive in its course, and usually terminates fatally. The cause is unknown. It is much more common in males than in females. Its occurrence in childhood is exceedingly rare.

The chief lesion is in the lymph nodes which become greatly enlarged, and besides new ones develop during the course of the disease. The spleen is usually, the liver less frequently, involved and somewhat enlarged by the formation of lymphomatous masses which may also infiltrate almost any tissue of the body. Microscopically, the early changes in the glands consist in an increase in the lymphoid tissue. Later there is proliferation of the endothelioid cells, the formation of giant cells, and an overgrowth of connective tissue. The eosinophile cells are frequently present in the tissues in great numbers. Any of the external or internal groups of lymph glands may be affected, and in severe cases the disease may involve almost every chain of glands in the body. Of the external groups, the cervical and the axillary are usually most affected; of the internal groups, those of the mediastinum and the retro-peritoneal region.

The disease develops very gradually, often insidiously. The external glandular swellings are usually the first noticed, but sometimes it is the anæmia which first attracts attention; occasionally it is the local symptoms resulting from the pressure of internal glands, which may give rise to œdema, pain, cough, or dyspnœa. The progress is generally slow but steady, and the glands may reach an immense size. The blood changes are inconstant. As a rule, there is a relative increase in the lymphocytes, while the total number of white cells is generally less than normal, although sometimes increased.

Treatment is very unsatisfactory. Arsenic in full doses appears to benefit some patients. The use of the X-ray has produced striking, though in most cases only temporary improvement in the external glands.



## CHAPTER III.

*DISEASES OF THE SPLEEN.*

**Weight.**—From 140 observations made at the New York Infant Asylum the following were the weights recorded at the different ages:

*Weight of the Spleen in Infancy and Early Childhood.*

| Age.               | Ounces.        | Grammes. |
|--------------------|----------------|----------|
| Birth.....         | $\frac{1}{4}$  | 7.7      |
| Three months.....  | $\frac{1}{2}$  | 15.5     |
| Twelve months..... | $\frac{3}{4}$  | 23.2     |
| Two years.....     | $1\frac{1}{4}$ | 38.5     |
| Three years.....   | $1\frac{1}{2}$ | 46.4     |

**Position and Methods of Examination.**—The normal position of the spleen is close against the diaphragm, its external surface being opposite the ninth, tenth, and eleventh ribs. Its anterior border comes as far forward as the middle axillary line, its posterior border being usually near the vertebral column. In infancy it is practically impossible to outline the spleen by percussion, unless it is enlarged. During full inspiration the spleen is often depressed enough to be felt at the free border of the ribs, but at other times it can not be felt unless it is enlarged or pushed downward by some pathological condition in the chest. Normally, the long axis of the spleen is nearly parallel with the ribs, but when the organ is much enlarged, its axis corresponds nearly with a line drawn from the axillary line at the border of the ribs to the middle of Poupart's ligament.

The thin abdominal walls of young children render palpation of the spleen much easier than in adults; and this is a much more satisfactory method of examination than is percussion. For satisfactory palpation it is necessary that the abdominal walls should not be tense. The child should lie upon his back with the thighs flexed and the skin, of course, bared. The physician, always having taken the trouble to warm his hands, should stand upon the left side of the patient and make pressure with the tips of the fingers, which are semi-flexed. The pressure should be at first light, and gradually increased, the fingers being then held stationary during two or three respiratory movements. Under ordinary conditions the spleen can easily be felt when it is sufficiently enlarged to be of any diagnostic importance.

When moderately enlarged, the lower border of the spleen is an inch or so below the free border of the ribs; when greatly enlarged, it forms a tumour which may nearly fill the left half of the abdomen. A tumour

in the left hypochondriac region is recognised to be the spleen, by the fact that it is freely movable laterally and at its lower border or extremity, while it is attached above; also its inner border can usually be felt to be thin and sharp, and marked about its middle by quite a deep notch.

#### ENLARGEMENT OF THE SPLEEN.

**In Acute Disease.**—The spleen is most frequently and most constantly enlarged in malarial and typhoid fevers, but it is occasionally so in all the acute infectious diseases.

In most of these cases the enlargement is chiefly from congestion, but there may be acute hyperplasia and an increase in size of the Malpighian bodies. It may contain small hæmorrhages, and in extremely rare cases the spleen may rupture. It is generally dark-coloured, soft, and somewhat friable. In the cases which recover, the splenic swelling subsides with the original disease.

**In Chronic Disease.**—Like the lymph nodes, the spleen is much more often enlarged in children, particularly young children, than in adults. Enlargement is seen at times in almost all the chronic diseases of early life; but it occurs most frequently in rickets, syphilis, malaria, tuberculosis, the blood diseases, and in amyloid degeneration. Besides, it may be the seat of a primary growth, either benign or malignant.

*Rickets.*—The splenic enlargement which accompanies rickets is generally seen during the first year; at this period it is very frequent. The swelling is usually moderate, but occasionally it is so great that the lower border is three or four inches below the ribs.

*Syphilis.*—Enlargement of the spleen is one of the most constant lesions of hereditary syphilis. It is present with great uniformity in children born with syphilitic lesions, and very frequently during the active period of the disease in early infancy. It is seen at a later period during infancy or childhood, associated with other late symptoms.

*Malaria.*—The swelling in cases of chronic malaria may be very great. The liver is not so often enlarged as in syphilis.

*Tuberculosis.*—It is rare to find anything more than a moderate swelling of the spleen in tuberculosis. In the most acute cases this may be due to the fever and general infection; in those which are less rapid, it depends either upon tuberculous deposits or passive congestion from venous obstruction.

*The Blood Diseases.*—Marked enlargement of the spleen is found in many cases of simple anæmia accompanied by moderate leucocytosis. The spleen is constantly swollen, and usually greatly so, in the pseudo-leukæmic anæmia of infants, in leukæmia, and in Hodgkin's disease. In the last two diseases the liver is also enlarged, but to a much less degree than the spleen; in the others it is but slightly changed.

*Amyloid Degeneration.*—The spleen is constantly involved in amyloid disease, and the enlargement of this organ, as well as that of the liver, may be very great.

*Cardiac Disease.*—In all forms of cardiac disease, and in other conditions in which there is obstruction to the systemic venous circulation, the spleen is enlarged. It is seen in congenital as well as in acquired cases. The liver is usually enlarged, and there may also be dropsy of the feet.

*New-growths, Tumours, etc.*—It is seldom in early life that the spleen is the seat of new-growths; these are usually varieties of sarcoma, but carcinoma has also been reported.

*Primary Spleno-megaly.*—The rare cases of immense primary enlargement of the spleen have been variously interpreted. By some writers the condition has been regarded as lymphoma. Bovaird<sup>1</sup> has reported two cases in children, sisters, one of which was carefully studied microscopically, and the conclusions reached that the process was an *endothelial hyperplasia*. The condition was first described by Gaucher. Clinically the disease is characterised by a slowly progressing enlargement of the spleen, which begins in early childhood and may continue for from five to twenty years; the size attained is very great, it often nearly filling the abdomen. In one of Bovaird's cases the weight was twelve and a half pounds. The other symptoms are a simple anæmia, inflammation of the gums with hæmorrhages from the nose, gums, and sometimes beneath the skin, and finally secondary symptoms due to the abdominal tumour. The course is very chronic, and thus far no known treatment has been of any avail.

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## CHAPTER IV.

### DISEASES OF THE BONES AND JOINTS.

#### ACUTE ARTHRITIS OF INFANTS.

THE terms *acute purulent synovitis*, *acute epiphysitis*, *pyæmia of bone*, and *acute osteomyelitis*, have all been applied to this condition. The disease is really a form of pyæmia. The causes and lesions may differ considerably in the different cases, but clinically they all have certain features in common, viz., an acute joint inflammation with supuration.

The acute arthritis of infants is essentially a disease of the first year, and is much more frequently seen in the first six months. The inflammation may begin in the joint, at the epiphyseal junction, or in the

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<sup>1</sup> American Journal of the Medical Sciences, October, 1900.

medullary canal; but, however it may start, the joint is soon invaded. The nature of the arthritis varies somewhat with the exciting cause. When it is due to the gonococcus, it is usually confined to the joint; there is in most cases a superficial inflammation involving the synovial membrane, but rarely leading to destructive changes in the cartilage, ligaments, or bone. When it is due to the streptococcus or staphylococcus, it may begin elsewhere than in the joint, which, however, is usually soon involved, and complete disorganisation may follow. It may also result in a diffuse osteomyelitis, in a subperiosteal abscess, or a separation of the epiphysis. As a late result there may be a pathological dislocation or a "flail joint"; less frequently there is ankylosis.

**Etiology.**—The cause of acute arthritis in infants is the entrance of pyogenic organisms into the circulation. In my experience the organism most frequently found is the gonococcus; next to this the streptococcus and staphylococcus; occasionally the pneumococcus, and very rarely the influenza bacillus. In most cases occurring during the first two months of life, the portal of entry is probably the umbilical cord. Less frequently infection takes place through the skin, conjunctiva, genital tract, or the mouth. In the cases developing later it is often difficult to determine the point of entry, especially when the cause is the gonococcus. During the last few years 26 cases of acute gonococcus arthritis have been observed in the Babies' Hospital, only 2 of which, occurring during the first month, could be classed as infections of the newly born. The cases were observed during a hospital epidemic of gonococcus vaginitis, and yet 19 were in male children, in no one of whom was there any genital lesion, and in only one was there conjunctivitis. Of the 7 cases occurring in girls, only 2 had vaginitis. The portal of entry in these cases could not be definitely determined.

**Symptoms.**—General symptoms often precede the local ones. In the most acute cases the temperature is high and widely fluctuating, accompanied by other symptoms of a severe infection. The earliest local symptoms are pain and tenderness, soon followed by swelling, which may develop quite rapidly in a single joint, or in several joints simultaneously. In those superficially situated there is redness of the skin, and fluctuation may be evident in three or four days. In cases coming on more gradually the temperature may be only from 100° to 102° F., and suppuration may not occur for two or three weeks. In the most severe cases the progress is rapid, one joint after another being involved, with general symptoms of pyæmia, and death may occur in a week or ten days, usually from some visceral inflammation, pneumonia, pericarditis, or meningitis. This very severe course is less frequent than the more protracted one where symptoms last from two to four weeks. Unless the pus is evacuated, extensive burrowing may take place.

In Townsend's collection of 73 cases, the joints were involved in the

following order: Hip, in 38; knee, in 27; shoulder, in 12; wrist, in 5; ankle, in 4; elbow, in 4; small joints, in 4. In three-fourths of these cases only a single joint was affected. In my own 26 gonococcus cases, the localisation was as follows: Finger or metacarpus, in 20; ankle, in 18; knee, in 17; wrist, in 12; toe or metatarsus, in 10; shoulder, in 9; elbow, in 5; temporo-maxillary, in 1; hip, in 1. The average number of joints involved was 4 or 5, the largest number being 8. The tendency of the gonococcus infections to involve the small joints is striking.

**Diagnosis.**—When several joints are involved, the disease is often mistaken for rheumatism, which, however, at this age is so rare that it may be ignored. Syphilitic epiphysitis resembles it in the localised tenderness and disability; but the rapid swelling and the severe constitutional symptoms are lacking.

**Treatment.**—Cold applications or wet dressings may be useful in relieving the symptoms. In some cases, most frequently when the cause is the gonococcus, the inflammation subsides without suppuration. In infections due to other organisms, suppuration almost invariably occurs and early free incision should be practised, followed by fixation of the joint. The results depend in no small degree upon the promptness with which the pus is evacuated. In the gonococcus cases there may be complete recovery. In most of the others the functions are impaired.

#### TUBERCULOUS DISEASE OF THE BONES AND JOINTS.

The chronic forms of tuberculous bone disease, on account of their insidious onset and the frequency with which they simulate other diseases, more frequently fall, in the early stage at least, into the hands of the physician than into those of the general or orthopædic surgeon. All that will be attempted in this chapter will be to outline in a general way the most important forms—viz., disease of the vertebræ, hip, and knee—dwelling particularly upon the early symptoms and diagnosis. For their fuller discussion, particularly as to the details of treatment, the reader is referred to text-books on general or orthopædic surgery. The causes are the same, and the lesions are very similar in all forms, and will therefore be considered together.

**Etiology.**—The age at which tuberculosis of the bones most frequently begins, is from the third to the eighth year, it being comparatively rare before the end of the second year. The sexes are affected with about equal frequency. Tuberculous bone disease may occur in a child who has previously been in apparent health, but more often in one who has been reduced by some previous illness, especially the infectious diseases; of these, it most frequently follows measles and whooping-cough. Of sixteen cases investigated by Park, the bacillus was of the human type in every instance.

A family history of tuberculosis is present in a large number, but by no means in a majority, of the cases. Like tuberculosis of the cervical glands, it is rarely preceded by other tuberculous processes, although it may be followed by them. It usually appears as an example of primary infection; but it seems very improbable that such should actually be the case. It is more likely that there has previously been a latent focus of tuberculosis elsewhere in the body. In many cases disease of the bronchial glands has been demonstrated by autopsy. Infection from these or from other tuberculous lymph glands is the most probable explanation of the origin of infection in cases of bone disease.

Traumatism is often an exciting cause, and it may determine the site of the disease.

**Lesions.**—The tuberculous joint diseases of childhood are, as a rule, secondary to disease of the bones. Hip-joint disease usually begins in the head of the femur, and knee-joint disease in one of the condyles; ankle-joint disease in the lower epiphysis of the tibia, etc.

The frequency with which disease is seen in the different locations is shown by the following table, which gives the number of cases of each form applying for treatment at the Hospital for Ruptured and Crippled, New York, during ten years:

|                      |                                |
|----------------------|--------------------------------|
| Spine.....           | 2,145 cases, or 37.5 per cent. |
| Hip.....             | 1,937 " " 34.0 " "             |
| Knee.....            | 1,222 " " 21.5 " "             |
| Ankle or tarsus..... | 255 " " 4.5 " "                |
| Elbow.....           | 71 " " 1.2 " "                 |
| Wrist.....           | 50 " " 0.9 " "                 |
| Shoulder.....        | 24 " " 0.4 " "                 |
| Total.....           | 5,704 100.0                    |

The character of the bone disease upon which chronic joint disease depends is generally a primary osteitis, which affects the articular extremities of the long bones, usually beginning near the epiphyseal line; in the short bones it is a central osteitis. The stages in the process are, first, congestion, swelling, and cell infiltration, followed by caseation, and frequently by softening and suppuration. In the early stage, the bone is slightly enlarged, and on section one or more yellowish foci of disease are seen. The disease may be arrested in this stage, encapsulation of the inflammatory products taking place; or it may continue until there is a more or less extensive breaking down or disintegration of the affected bone. As the disease extends there are involved the periosteum, the articular cartilage, and finally the joint itself. Abscess may form in the joint or in the soft parts surrounding the bone. The process is quite analogous to tuberculous disease of the lung. As the disease advances ligamentous attachments are loosened, and displacement of the parts

occurs with the production of deformity, due partly to muscular contraction and partly to the weight of the body. The inflammatory process, with its resulting disintegration, generally goes on to a certain point, where it is arrested. Gradually the broken-down bone substance is separated and thrown off in small particles in the discharge, and a reparative process begins, with the formation of healthy bone. Where joint structures have been destroyed, cure takes place by bony ankylosis. Sometimes the disease finds its way to the surface without involving the joint; at other times the disease may be arrested, and its products become encapsulated within the bone. Inflammation of the joint may occur by a gradual extension of the inflammatory process, or by a sudden perforation of the articular lamella. As a result of extensive disease, all the joint structures may be affected—the synovial membrane, ligaments, articular cartilages, and the cellular tissue surrounding the joint. The process of disintegration and that of repair are both very chronic and measured by months or years. The entire course of the disease is from one to ten years, three years being about the average duration. In the great proportion of cases but one joint is involved, although it is not infrequent in hospitals to see two, three, and sometimes four of the large joints affected in the same patient.

*Secondary Lesions.*—Abscesses form in a considerable proportion of the cases, and often burrow a long distance before they reach the surface. Amyloid degeneration of the liver, spleen, and kidney, and sometimes of the intestines, occurs as the result of the prolonged suppuration, chiefly in connection with disease of the hip or spine, occasionally with that of the knee. General or localised tuberculosis, particularly tuberculous meningitis, may develop at any time and prove fatal.

*Caries of the Spine—Pott's Disease.*

This consists in a tuberculous inflammation of the bodies of the vertebræ, usually beginning in the central portion and extending to the periosteum, ligaments, cartilages, and, in fact, to all the contiguous structures. Secondly it involves the membranes of the cord, the roots of the spinal nerves, and even the cord itself. The number of vertebræ usually affected is from two to five. The gross appearance of the lesion in a well-marked case is shown in the accompanying cut (Fig. 165). After the bodies of the vertebræ have become softened and partially broken down by disease, the pressure from the superincumbent weight of the body causes them to fall together and produces a backward displacement of the spinous processes, giving rise to the deformity known as kyphosis, which in its extreme form is popularly known as "hunchback."

Any part of the vertebral column may be affected; but the disease is most frequent in the dorsal region, as shown by the following statistics

from the Hospital for Ruptured and Crippled: Of 2,143 cases, 72.5 per cent affected the dorsal region, 15.3 per cent the lumbar region, and 12.2 per cent the cervical region.

**Symptoms.**—The onset is gradual, often insidious, and the early symptoms are frequently overlooked or misinterpreted. The case may go on for weeks or even months before the true nature of the disease is recognised, which is often not until deformity has occurred. In nearly all cases, however, the early symptoms are sufficiently characteristic to enable a careful observer to make a diagnosis before the stage of deformity.

The most constant early symptoms are: (1) Pains caused by the irritation of the nerve roots and referred to various parts of the body, following the distribution of the spinal nerves; (2) rigidity of the spine from muscular spasm, this being an attempt to prevent motion at the seat of disease; and (3) the assumption of various postures calculated to relieve pressure upon the diseased vertebral bodies. Sometimes the first symptoms are those of pressure-paralysis; at others they are the local signs of abscess. In addition to the local symptoms mentioned, there is usually disturbed sleep, often accompanied by moaning.

**Cervical Disease.**—The pains are often felt above the point of disease, frequently in the form of occipital neuralgia; sometimes they are referred to the front or the side of the neck. They may be so frequent and so severe that the face assumes a constant expression of anxiety or distress. In other cases pain is excited only by an attempt at movement. The muscular spasm most frequently takes the form of slight torticollis, sometimes of slight opisthotonus; sometimes there is simply a fixation of the head by a tonic spasm of all the muscles of the neck; both active and passive motion is resisted, and any movement may be so painful that the child involuntarily steadies his head with his hands. These symptoms come on gradually and are persistent. Sometimes they are overlooked, and the first thing to attract attention is a progressive weakness in the lower extremities, which proves to be the beginning of paraplegia. Occasionally the first marked symptoms are those due to the formation of a retro-pharyngeal or a retro-oesophageal abscess.

The deformity from cervical disease develops much later than when

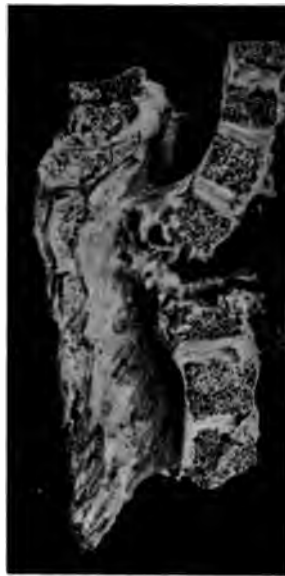


FIG. 165.—POTT'S DISEASE OF THE UPPER DORSAL REGION. A vertical section of the spine, showing disintegration of the bodies of the vertebrae and encroachment upon the spinal canal. (From a patient dying in the Hospital for Ruptured and Crippled.)



the disease is located elsewhere. Usually the neck appears **broadened** or thickened in a nearly uniform way, and often the head seems to have settled downward upon the shoulders. In the lower cervical region a **kyphosis** is not infrequent; but in the middle and upper regions there is more often an anterior prominence, which may be felt in the posterior wall of the pharynx.

*Dorsal Disease.*—The referred pains are now below the seat of disease, and take the form of intercostal neuralgia or pain in the epigastrium or the abdomen. They are often ascribed to cold, malaria, indigestion, or worms. There is a disposition to assume the prone position while sleeping, and also to lean across a chair or the lap of the nurse. The child walks carefully, holding the spine erect and very stiffly, and exhibits great caution in getting into or out of bed, or in rising from a recumbent position. In the beginning there may be a slight lordosis, or forward curve at the seat of disease, instead of the usual kyphosis or backward projection, but the latter soon takes its place, and with it is seen the compensatory lordosis in the lumbar region.

*Lumbar Disease.*—The first symptoms here are often pain and lameness, referred to one of the lower extremities. This frequently leads to the suspicion that the hip is the seat of disease. In addition to the lameness there may be a tilting of the pelvis to one side, and sometimes quite a distinct lateral curvature of the spine. Referred pains are not so frequent nor so severe as when the upper part of the spine is affected; they may be felt in the groin, in the loin, in the thigh, in the buttock, or in the hypogastrium. The gait and attitude are very characteristic: Throwing the shoulders well back, the patient walks stiffly, with short steps, holding the spine with the greatest care. He rises from the floor awkwardly and with difficulty. Deformity is not usually so early or so marked as when the disease is dorsal, and often before it is visible there are symptoms due to the formation of psoas abscess—lameness, flexion of one thigh, and a tumour deep in the iliac fossa or at the upper and inner aspect of the thigh; in both locations it has often been mistaken for hernia.

*Physical Examination.*—Whenever any of the above symptoms are present, the child should be stripped and submitted to a thorough examination, the purpose of which should be to determine, first, the existence of any deformity; secondly, the mobility of the spine; thirdly, the presence of any secondary lesions, such as abscesses or paralysis. The mobility of the spine is best determined by studying the attitude, gait, and posture of the child, and the manner of stooping or rising from the floor. The gait has already been described with the symptoms of lumbar disease. As it has been aptly put, "the child walks with his legs, but not with his back." In stooping, the same disinclination to bend or move the spine is seen. It is often impossible to induce the child to

stoop at all, and when he does so, to pick up some object, there is acute flexion at the knee and hip, but as little bending of the spine as possible. In rising from the recumbent position the same thing is seen. The posture and attitude of the child will be modified by the position of the disease, and somewhat by the activity of the process at the time; however, by comparing the movements referred to with those of a healthy child, the great difference will at once be apparent. If the symptoms point to cervical disease, a digital exploration of the pharynx for deformity or abscess should be made, and the extremities should be examined for paralysis. If the disease is in the lumbar region, deep palpation of the iliac fossa should be made to discover a psoas abscess, and the passive movements of the thigh should be carefully tested to determine whether there is any resistance to extreme extension, this often being present before the psoas tumour. No matter how clearly the lameness may be at the hip, it should be remembered that this often results from disease of the lumbar spine. If the thigh is flexed and freely movable except in extension, the symptoms are probably the result of psoas irritation, for in hip-joint disease the other movements of the joint are also resisted.

The deformity of Pott's disease is often spoken of as "angular" curvature of the spine. While this is a true description of the disease at an advanced stage, there is often in the early stage only a general curve. Later a slight knuckle is seen from the unnatural projection of a single spinous process. This deformity may increase and finally involve five or six vertebrae. It is usually greatest in the upper dorsal region. A slight prominence, which does not disappear on suspending the patient, is always suspicious.

Tenderness upon pressure over the spinous processes and increased sensitiveness to heat and cold are rarely present. Pain may sometimes be produced by downward pressure upon the head or shoulders in the axis of the spine. This symptom is not necessary for diagnosis, and the attempt to elicit it is strongly condemned by Gibney, who has seen serious harm follow such a test.

*Course of the Disease.*—Caries of the spine is a very chronic disease, its course being measured by months or years, but marked, as in all chronic diseases, by periods of remission and exacerbation. An exacerbation may follow traumatism, and is often accompanied by the formation of an abscess. After the disease has lasted from one to three years, the destructive inflammation usually ceases and repair begins, a cure being finally effected by a process of consolidation of the fragments of the diseased vertebrae, and the production of ankylosis. Relapses are easily excited by traumatism, by improper treatment, or by discontinuing the use of mechanical supports before the disease is arrested.

**Abscesses.**—The frequency with which abscesses occur depends somewhat upon the treatment. Townsend states that of 380 cases, abscess was present in twenty per cent. They are rarely seen earlier than three or four months from the beginning of symptoms, and usually belong to the second year of the disease. They sometimes form with acute symptoms, but more frequently they appear as typical cold abscesses. Those connected with cervical disease are retro-pharyngeal or retro-oesophageal, or they may open externally, usually just above the clavicle, in front of the sterno-mastoid muscle. Those with disease of the lower cervical and upper dorsal vertebræ are apt to burrow along the spine, appearing in the lumbar region; rarely they may rupture into the oesophagus or the pleural cavity. Those with disease of the lower dorsal or lumbar vertebræ may open just above the iliac crest posteriorly, or burrow anteriorly between the abdominal muscles, but the usual course is for them to follow the psoas muscle, appearing in the groin just above Poupart's ligament or at the upper and inner aspect of the thigh.

Paralysis occurs in about one-half the cases in which the disease affects the lower cervical and upper dorsal vertebræ, but it is rare when the disease is below the middle dorsal region (see Compression Myelitis).

**Prognosis.**—The actual mortality of Pott's disease is difficult to state, so many of the consequences of the disease being remote and not fully appreciated until adult life is reached. The general mortality from all causes is from ten to twenty per cent. The causes of death are exhaustion from prolonged suppuration, amyloid degeneration, myelitis, general tuberculosis, and tuberculous meningitis. Sudden death occasionally occurs from pressure upon the cord in the upper cervical region, or from the pressure effects of abscesses in the posterior pharynx or in the posterior mediastinum.

The prognosis as to the amount of permanent deformity will depend upon the seat of the disease, the time at which treatment is begun, and upon the thoroughness with which it is carried out. The best results as to deformity are obtained when the disease is below the middle dorsal region. With improved methods of treatment begun early, a large number of these patients recover with an insignificant amount of deformity, and some with none whatever.

**Diagnosis.**—The spinal deformity resulting from Pott's disease may be confounded with rachitic kyphosis or with rotary lateral curvature. Rachitic curvatures are usually seen in children under eighteen months of age, a time when Pott's disease is rare; there are other signs of rickets present, and instead of rigidity there is usually undue mobility of the spine. What is true of rickets may be said of all curvatures depending upon malnutrition. Rotary lateral curvature is seen about puberty, rarely in young children except in connection with rickets. A slight lateral deviation of the spine, sometimes seen in the early stages of caries,

may resemble a case of incipient rotary curvature. The latter is not attended by pain or rigidity, and is most frequent in young girls from eleven to fourteen years of age.

Other abscesses may be mistaken for those dependent upon vertebral caries. This difficulty is likely to exist in the cases attended by very little spinal deformity. These abscesses are most frequently in the iliac fossa or in the lumbar region, and may be due to perinephritis or appendicitis. The latter are more acute than those depending upon bone disease and usually accompanied by fever. Tumours of the vertebræ or of the spinal cord may give rise to symptoms almost identical with those resulting from compression myelitis due to Pott's disease, but both of these are extremely rare.

**Treatment.**—The treatment of Pott's disease is both general and local, and neither should be neglected. The constitutional treatment should be similar to that employed in other forms of tuberculosis.

The indications for local treatment are to put the diseased parts at rest, by immobilising the spine and removing the superincumbent weight of the body. With the great advances made in orthopaedic surgery it is no longer necessary to confine these patients in bed, as was formerly practised, to secure this result. It may be accomplished either by plaster-of-Paris, or some other form of jacket, or a properly fitting steel brace. A head-support should be attached to all forms of apparatus, if the disease is above the middle dorsal region. The closest attention to details and much experience in the use of apparatus are required to secure the best results. In perhaps no class of cases have the beneficial results of modern scientific treatment been more apparent than in those of Pott's disease. For the details in regard to the mechanical treatment and the different forms of apparatus, the reader is referred to works on general or orthopaedic surgery.

#### *Articular Ostitis of the Hip—Hip-Joint Disease.*

In early childhood this generally begins as a chronic ostitis in the head of the femur, starting near the epiphyseal line. Exceptionally, and oftener in older children, it begins in the acetabulum. The pathological process, as well as the clinical history, is generally described as consisting of three stages. In the first stage—that of ostitis—the lesions are limited to the bone; in the second stage—that of arthritis—all the joint structures are involved, and in this stage suppuration usually occurs; in the third stage there is breaking down and absorption of the head and sometimes of the neck of the femur, which, with destruction of the ligaments, leads to marked displacement of the parts from muscular contraction. The disease may be arrested in the first or in the second stage, or it may continue through all three stages.

**Symptoms.**—Clinically, the usual duration of *the first stage* is three or four months; it may last only for a few weeks, it may extend over two or three years, and the disease may be arrested in this stage. The onset is usually very gradual, and the symptoms are often considered of trivial importance until they have continued for some weeks. Generally the first thing noticed is slight lameness, due to stiffness of the joint. In the beginning this may be seen only in the morning, wearing off during the day. It may be accompanied by some tenderness about the hip and a disinclination to walk. A little later the child complains of pain, which is most frequently referred to the front of the knee or the inner aspect of the thigh, but only in rare cases to the hip itself. This is slight at first, but gradually increases in frequency and severity, and soon there are added the “starting pains” at night, which are one of the most characteristic features of early hip disease. These pains are produced by a sudden spasm of the muscles during sleep. The child often cries out sharply without waking, sometimes wakes with a cry; this is often repeated several times during the night. Soon restlessness and fretfulness during the day are present. The lameness, which at first was slight and occasional, or noticed only in the morning, comes to be a constant symptom, and week by week increases in severity. The evolution of these symptoms may take only a few weeks, but sometimes they come and go in the most inexplicable manner during a period of several months, or even one to two years, before they are fully developed.

Every child with a suspicious lameness, or with pains like those mentioned, should be stripped and submitted to a thorough examination. The first points to be observed on inspection relate to the general contour of the hip; every prominence and depression should be carefully noted. Then the attitude and gait should be studied; and finally all the functions of the joint should be carefully tested, and the limbs measured, to determine the existence of shortening or atrophy. At every step a comparison should be made with the sound limb. The contour of the hip is changed quite uniformly; there is broadening and flattening of the whole gluteal region; the trochanter is unnaturally prominent; the gluteal fold is shortened, and often single instead of double. There is no characteristic position of the limb in this stage. There is atrophy of the thigh and often of the calf. In Fig. 166 is shown the appearance of a typical case in the full development of the first stage. In walking, the child favours the diseased side, throwing the weight as much as possible upon the sound limb; but all these symptoms are of much less importance for diagnosis than is an examination of the functions of the joint.

For this purpose the child should be placed upon a table upon his back, and the various movements of the hip—abduction, adduction, flexion, extension, and rotation—should be executed, first with the

sound limb and then with the suspected one, the two being carefully compared at every point to determine the degree of motion allowed. It is not necessary that force should be employed or pain inflicted. If the symptoms have existed for some weeks, there is generally a limitation of motion at the hip in all directions, but first usually in abduction, rotation, or extension. In more advanced cases, no motion whatever may be permitted at the joint, the pelvis tilting with the slightest movement of the femur. This fixation of the hip is due to tonic muscular spasm. Crowding the articular surfaces together, by pressure upon the heel or trochanter, produces pain, which is usually referred to the joint. This test should be carefully made, lest injury be inflicted. Gibney cautions against examinations under ether, since in this way serious injury may be done unconsciously.

*Second Stage.*—This has been called the stage of arthritis. Its existence may be assumed when the limb takes the position of marked permanent deformity, which is due at this period to muscular action, not to destructive bone changes. The transition from the first to the second stage is in most cases a gradual one, and the line between the two can not be sharply drawn; sometimes, however, it is rapid, and marked by a sharp exacerbation of all the symptoms. This may indicate a sudden perforation of the joint and the rapid development of suppurative arthritis. Such is the usual result when an abscess which has been slowly forming in the bone opens into the joint; or acute joint inflammation may be lighted up without so evident a cause. Sometimes the pus reaches the surface below the capsular ligament, and the joint remains intact. An acute exacerbation is indicated by increased pain, excessive tenderness about the hip, often by inability to walk, or even to bear any weight upon the limb, and frequently by fever. The position assumed by the limb is now fairly characteristic. The foot is generally everted, the thigh slightly flexed and rotated outward, and the limb apparently lengthened. There may be infiltration anywhere about the hip, due to the formation of an abscess. The muscular spasm is so great that the joint is locked—no motion whatever being allowed. Abscesses may form



FIG. 166.—HIP-JOINT DISEASE, AT THE END OF THE FIRST STAGE. Showing muscular atrophy, prominence of the trochanter, flattening of the gluteal region, and a single gluteal fold.

at any point about the hip; they are especially frequent at the upper and outer aspect of the thigh, and may burrow long distances before reaching the surface. The duration of the second stage also is indefinite, but it usually lasts from a few months to a year, or the disease may be arrested in this stage.

**Third Stage.**—There is now marked deformity, which is the result of muscular contraction after absorption of the head and sometimes the neck of the femur, and destruction of the ligaments. The position of the limb is a very constant one, and resembles that present in dislocation upon the dorsum of the ilium. There is shortening of from one to four inches; the thigh is strongly flexed, adducted, and rotated inward, and the foot is inverted; the trochanter lies against the outer surface of the ilium, and is above Nélaton's line. In this position the joint may become ankylosed. The displacement usually comes on gradually, but it is sometimes so sudden as to be mistaken for a true dislocation, although the latter is exceedingly rare in the course of hip disease.

There is now marked atrophy of all the muscles of the limb, and the thigh may be two or three inches smaller than its fellow. No motion at all is usually allowed at the hip, but this is compensated for to some degree by the exaggerated mobility of the lumbar spine. The spinal curvature—lordosis—is very marked both upon standing and walking. The duration of this stage may be several years. From time to time exacerbations occur, often excited by falls, and accompanied by the formation of new abscesses. In protracted cases, all the soft parts about the hip may be seamed with cicatrices from old sinuses. After the disease has gone on to the third stage, cure can take place only by ankylosis.

**Diagnosis.**—The important point in the early diagnosis of ostitis of the hip, is the gradual evolution of the symptoms, the most characteristic of which are lameness, "starting pains" at night, and impairment of all the functions of the joint. Mistakes in diagnosis most frequently arise from a failure to obtain a careful history, and from relying too much upon the symptoms of lameness and deformity. The essentially chronic character of the disease should constantly be borne in mind. In the vast majority of cases, with a careful history and a thorough examination, there can be but little doubt as to the diagnosis except at the very outset. The proportion of obscure and irregular cases to those following the regular course is small.

In the early stage, hip-joint disease may be confounded with a strain of the joint, with muscular rheumatism, poliomyelitis, periostitis of the shaft of the femur, phlegmonous inflammation in the neighbourhood of the joint, or with caries of the lumbar spine. In the second stage there is even less difficulty in diagnosis, although abscesses resulting from perinephritis or appendicitis have been mistaken for those arising from hip disease. In the third stage, a mistake is almost impossible.

**Prognosis.**—This is to be considered both with reference to life and limb. The records of the Hospital for Ruptured and Crippled show the mortality of hospital patients with hip disease to be nearly twenty-five per cent. This includes deaths directly or indirectly traceable to the disease. The causes are nearly the same as in caries of the spine—exhaustion from prolonged suppuration, amyloid degeneration, and general tuberculosis or tuberculous meningitis.

Under the most favourable conditions, the disease may be arrested in the first stage, and recovery occur without lameness or any noticeable impairment of the joint functions. This result, however, is not often obtained, because the disease is usually well advanced before it is recognised, or because of the difficulty in the way of carrying out all the details of treatment in the best possible manner. If the disease has advanced to the second stage and suppuration has occurred, there always results some impairment of the joint functions; usually there are decided lameness and marked muscular atrophy, but very little shortening or deformity, provided the limb has been kept in the proper position. If the disease has advanced to the third stage, there are always marked shortening, deformity, and lameness.

**Treatment.**—The indications for constitutional treatment are the same as in caries of the spine. The purpose of local treatment is to secure constant and complete rest for the diseased parts, and to prevent deformity. Rest is secured by overcoming the muscular spasm by means of extension, by immobilising the joint, and by transferring the weight of the body, in walking, from the hip to the perinaum. All these indications are now met, while the patient is up and about, by the use of the most approved apparatus. The general opinion of orthopaedic surgeons at the present day is against excision, except in cases where, in spite of treatment by apparatus, the disease has advanced to the third stage, and in cases where life is threatened from prolonged suppuration and exhaustion.

*Articular Ostitis of the Knee—Knee-Joint Disease—White Swelling.*

Ostitis of the knee usually begins in one of the condyles of the femur, the inner much oftener than the outer one; less frequently it begins in the head of the tibia. The pathological process is very much like that at the hip. There is in the first stage a central ostitis accompanied by infiltration and expansion of the part of the bone affected. The disease may remain limited to the bone, the inflammatory products becoming encapsulated, or softening and breaking down may occur, with the formation of an abscess. Gradually the process extends outward, and the periosteum and the soft parts are involved. The disease may invade the joint itself in a destructive inflammation; or pus may escape externally without seriously involving the joint structures. The degree to which



the joint is involved varies much in different cases; there may be only a simple synovitis, a suppurative arthritis, or a destruction of the cartilages and articular ends of the bones, synovial membrane, and ligaments, so that in the advanced stage all traces of a joint structure are lost.

If the process remains limited to the bone, recovery may take place with very little impairment of the joint functions. If suppuration in the joint has taken place, there will be more or less stiffness and fibrous or bony ankylosis. When there is destruction of the ligaments and articular ends of the bones, the limb assumes a characteristic position—the joint is flexed, the tibia is displaced backward and rotated outward, and there is marked over-riding of the femur. Bony ankylosis in this position is often seen.

**Symptoms.**—The earliest symptoms of disease at the knee are usually a slight stiffness of the joint, with a disposition to flexion and slight lameness. At first these symptoms are noticed only occasionally; finally they become constant and there is pain, which is usually referred to the knee. In some cases there are “starting pains” at night, although these are less constant and less severe than in hip disease. Swelling is noticed early, as the diseased parts are superficial. At first this is chiefly of the bone itself; the condyle, usually the inner one, is enlarged and elongated, often to a marked degree, before there is any infiltration of the soft parts. Later there is a general fusiform swelling, involving the entire joint and effacing all the normal outlines. Some tenderness upon pressure over the bone affected is present quite early, and there may be atrophy of the muscles of the thigh and calf. The knee is flexed and slightly rotated outward, the position which secures the most complete relaxation of the joint structures. In some cases there is seen the characteristic swelling due to distention of the synovial membrane. Abscesses may form anywhere about the joint; very frequently they burrow beneath the tendon of the quadriceps extensor as far as the middle of the thigh. Gradually the deformity increases until the leg may be flexed at a right angle, and rotated outward over an arc of twenty or thirty degrees.

The course of the disease resembles that of osteitis of the hip and the spine. During periods of remission pain and tenderness often subside for several months so completely as to lead to the supposition that the disease has been arrested. An exacerbation is often excited by a fall or a strain of the joint, or it may follow an attack of acute illness. The disease may then progress rapidly and abscess after abscess form, with extensive destruction of all the joint structures and the production of permanent deformity.

**Prognosis.**—The danger to life is considerably less than in disease of the hip or spine. Death, however, results from the same causes—exhaus-

tion, amyloid degeneration, and general tuberculosis or tuberculous meningitis.

With an early diagnosis and proper treatment the disease may, in a considerable proportion of cases, remain limited to the bone, and the resulting lameness and deformity be very slight; but otherwise a certain amount of lameness results from the stiffness of the joint. This may be due either to fibrous thickening or to bony ankylosis. Nearly all patients are able to walk without crutches, and if proper treatment has been carried out there is neither marked shortening nor deformity, although there is always great muscular atrophy.

**Diagnosis.**—The important symptoms for diagnosis, are the gradual onset, the early swelling which is due to enlargement of the bone, and the constant lameness and deformity. The disease may be confounded with rheumatism, with synovitis, and even with scurvy. In all these cases the resemblance exists only during the period of exacerbation. A careful history, however, will usually clear up the diagnosis.

**Treatment.**—The general treatment is the same as in other forms of joint disease. The indications for local treatment are the same as in hip disease—viz., to immobilise the affected limb and prevent deformity. This is accomplished by a form of apparatus which transfers the weight of the body from the joint to the perinæum, and which overcomes the muscular spasm which produces flexion and inward rotation of the joint. As in hip disease, the results with mechanical and constitutional treatment are decidedly better than from early operative measures; but late operations are indicated under the same conditions.

#### *Tuberculous Osteo-Myelitis.*

This disease is rarely seen except in the short tubular bones, most frequently those of the hand and fingers. From this fact it is often called *scrofulous* or *tuberculous dactylitis*. It is described by many writers under the name of *spina ventosa*. Unger gives the following figures showing the frequency with which the different bones were affected: Fingers in 43, toes in 3, metacarpus in 41, metatarsus in 14, radius in 2, ulna in 2, tibia in 3, jaw in 3. The first phalanx of the index finger is the bone which is most frequently the seat of disease. In the majority of cases the process is confined to a single bone, although it is not rare to see five or six affected. In such cases the disease is seldom symmetrical. The process is a chronic inflammation, beginning in the centre of the bone with the deposit of tuberculous material. The swelling which follows causes an expansion of the bone and thinning of the shaft, until a mere shell may remain. The later changes are inflammation of the periosteum and the soft parts, the formation of abscesses and sinuses, necrosis, the exfoliation of sequestra, etc. The entire disease lasts from one to three years, and causes in most cases marked deformity.

Tuberculous dactylitis is essentially a disease of early childhood, being seen most frequently during the second and third years. In a considerable proportion of the cases there is a family history of tuberculosis. The disease frequently appears to be the only tuberculous lesion in the body, but tuberculosis of the hip, knee, ankle, or spine may be associated.

**Symptoms.**—Tuberculous dactylitis usually begins as a painless enlargement of one of the phalanges, most frequently the first one of the index finger. It may be two or three months before it is of sufficient



FIG. 167.—TUBERCULOUS DACTYLITIS OF THE FIRST PHALANX OF THE INDEX FINGER.

size to attract much attention. Exceptionally the inflammation is a more active one, and is accompanied by both pain and tenderness. The swelling is quite characteristic; it is smooth, hard, uniform, and generally spindle-shaped, involving the entire phalanx of the affected finger. The appearance of a severe typical case is shown in Fig. 167. Later there is discolouration of the skin, and usually there is suppuration. The abscess generally opens at the side of the finger, and a curdy pus is evacuated. If the opening is enlarged by an incision there is found a cavity partly filled with caseous matter, and dead bone is felt, and perhaps a loose sequestrum. The cavity is surrounded by a thin shell of new bone, which is formed from the periosteum. If no operation is done the discharge continues for weeks or months, other abscesses often form, and finally several small sequestra are exfoliated—sometimes a single large one, which is the shell of the diseased phalanx almost entire.

In some cases the disease is arrested before necrosis occurs, but in the majority this is not so. After the wounds have all healed the finger

remains shortened, deformed, and often useless. In some cases the disorganisation is so extensive that amputation is necessary.

**Diagnosis.**—The recognition of dactylitis is usually easy, but as symptoms almost identical may be seen in a syphilitic inflammation, it is often difficult to tell with which of the two forms one has to deal. The tuberculous form is very much more frequent; it may occur in a patient with tuberculous antecedents, or it may be associated with other tuberculous lesions. Syphilitic cases are distinguished by the fact that the lesion is more frequently multiple, that it is often symmetrical, and that other manifestations of syphilis are generally present. The Wassermann and the tuberculin tests give definite information in nearly all cases.

**Treatment.**—Painting with iodine and like measures are useless. The diseased part should be kept at rest—if a finger, by the application of a splint. Every means should be taken to build up the patient's general health, as this is the most effective way to influence the local process. The general verdict of surgeons is against early excision as a means of arresting the disease. Abscesses should be opened early and freely, all diseased bone removed, the finger kept in proper position, and the wound treated according to general surgical principles. Under almost any treatment the disease is a protracted one, and rarely lasts less than a year.

#### SYPHILITIC DISEASES OF THE BONES.

The bone lesions of hereditary syphilis are not infrequent, but were long unrecognised. They may be divided into two groups—those occurring with the early symptoms, and those which belong to the late manifestations of the disease.

##### *Acute Epiphysitis.*

This is the most frequent variety of bone disease in early hereditary syphilis. It may begin even in intra-uterine life, and it forms one of the most characteristic lesions of the disease. To some degree it is almost invariably present in syphilitic foetuses and in syphilitic infants who are still-born.

In the early stage, there is an increase in the cartilage cells and often increased calcification. Later, a line of softening forms at the epiphyseal junction, which may cause loosening of the cartilages and ultimately complete separation of the epiphysis from the shaft, by the formation of granulation tissue between them. In cases receiving proper treatment, recovery may take place with good union, perfect function, and without any deformity. In other cases degenerative changes continue, and infection with pyogenic germs may be added. The large joints are usually affected, and the lesions are frequently symmetrical. Acute suppurative arthritis may occur independently of changes at the epiphysis; but even when these

are seen in syphilitic infants they are to be regarded as of **pyæmic** rather than of syphilitic origin. Secondary to the changes at the epiphysis, there is periostitis. Periostitis of the shaft is rare in early infancy.

The bones most frequently the seat of acute epiphysitis are the humerus, radius, and ulna, although any of the long bones may be affected.

**Symptoms.**—The early symptoms are usually quite acute, and appear during the first six weeks of life; they may precede any other mani-

festations of syphilis. In some cases there is first noticed an inability on the part of the child to move the limb, which may easily be mistaken for paralysis. It is, in fact, often described as "syphilitic pseudo-paralysis." The limb lies perfectly motionless, and any attempt at passive movement causes evident pain. There is tenderness on pressure, and soon swelling is seen, both being most marked at the epiphyseal line. If the bone affected is superficially situated, as the lower epiphysis of the humerus, radius, or tibia, swelling is very apparent, while it may be scarcely perceptible at the upper epiphysis of the humerus. The swelling is usually cylindrical and moderate in degree, being limited to the extremity of the bone. Separation of the epiphysis may take place, so that crepitation is obtained by moving the limb.

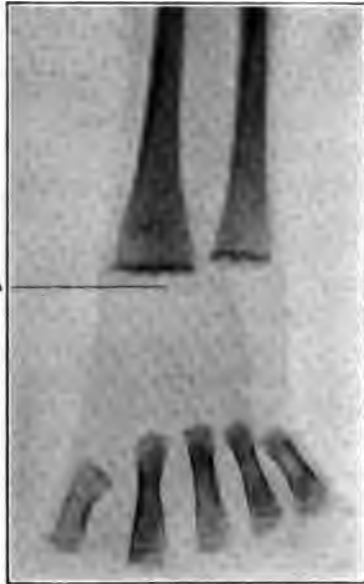


FIG. 168 — HEREDITARY SYPHILIS. Showing Gyron's line, A. Infant two months old.

With this there is sometimes suppuration. The Röntgen ray shows in many instances an increase in calcification at the epiphysis with an irregular serrated outline (Fig. 168) known as *Gyron's line*.

In the milder cases, or those which have been subjected to active treatment, both the swelling and the tenderness subside rapidly without suppuration; and even though the epiphysis has separated from the shaft, it speedily unites. When pseudo-paralysis has been the chief symptom, very rapid improvement occurs under treatment, and usually there is complete recovery of function in two or three weeks. If the disease extends to the joint, or if osteo-myelitis develops, the case is almost certainly fatal.

**Diagnosis.**—This is usually easy, from the age of the patient—generally under three months—the early prominence of pain and apparent loss of power, with the later appearance of swelling and signs of inflammation at the epiphyseal junction. In all these respects the disease

closely resembles scurvy; but the latter is rare before the eighth or tenth month; there is usually a history of the long-continued use of some proprietary infant food, and it is cured by dietetic treatment alone. In case of doubt the Wassermann test may be used.

The apparent loss of power may lead to the diagnosis of birth palsy, especially of the upper-arm type. The presence of acute pain and tenderness, the absence of the characteristic deformity, and the prompt recovery under constitutional treatment, usually make the distinction between the two conditions an easy one.

**Treatment.**—This is the same as in all early syphilitic manifestations, for which see the article on Syphilis. Locally, the part requires in the early stage only protection and rest. Should suppuration occur in the neighbouring joint, or should osteo-myelitis develop, these conditions should be treated surgically, as they are when due to other causes.

#### *Chronic Osteo-Periostitis.*

This is the usual form of bone disease which is seen in late hereditary syphilis, and it is one of the most frequent and most characteristic

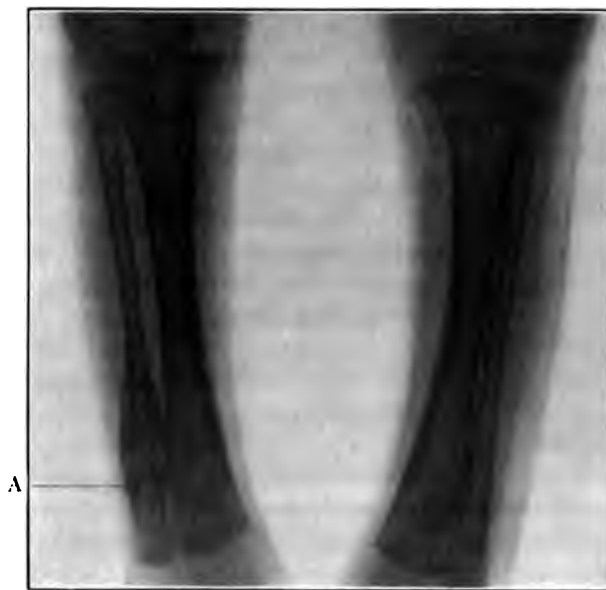


FIG. 169.—SYPHILITIC PERIOSTITIS OF THE FIBULA. Infant three months old. Same patient as Figs. 173-176. Right side affected; left side normal.

lesions of that stage of the disease. It is occasionally seen in early infancy, and usually affects the long bones. The lesions are multiple, and at this age principally periosteal. The Röntgen ray picture shows a fusiform swelling chiefly due to periosteal thickening (Fig. 169).

Chronic osteo-periostitis is more frequent after the third year, and most of the cases occur between the fifth and fourteenth years. The most frequent seat of disease is the tibia, and next to this the bones of the forearm and the cranium. The following is the frequency with which the different bones were affected in the series of cases reported by Fournier: tibia in 91 cases, ulna in 22, radius in 15, cranium in 16, humerus in 12, all others in 37. The process may result either in a diffuse or a localised hyperplasia of bone or in necrosis.

The typical changes are seen in the tibia. The shaft of the bone is principally or solely affected. There is often produced a very characteristic deformity, consisting of a forward curve of the anterior border of the tibia, which has been compared to a sabre blade (Fig. 170, 171).



FIG. 170.—SYPHILITIC OSTEO-PERIOSTITIS OF THE TIBIA. Left tibia greatly enlarged;  $1\frac{1}{2}$  inches longer than the right, and leg 2 inches larger in circumference; sabre-like anterior border. Right tibia normal; lesion of long standing. Patient 13 years old.

In some cases the bone is bent inward at its lower third, resembling somewhat a rachitic curvature. Sometimes the entire shaft of the bone is affected, and it may be greatly enlarged. At other times the swelling is chiefly near the epiphysis, where large bosses may form of sufficient size to interfere with the functions of the joint. Instead of affecting the bone uniformly, the disease often affects only certain

parts, leading to the formation of large nodes which are more likely to be followed by necrosis than are the other lesions. In most of the cases the process is purely a hyperplastic one, leaving the bone permanently enlarged and the limb often lengthened. Less frequently, there occur



FIG. 171.—SYPHILITIC OSTEO-PERIOSTITIS OF THE LEFT TIBIA.  
Similar lesion to that shown in Fig. 170; patient 8 years old. The right tibia is normal.

gummatous deposits in or beneath the periosteum, which may soften, suppurate, and lead to superficial necrosis, with the formation of sinuses that remain open until the sequestrum is exfoliated. Syphilitic deposits sometimes take place in the interior of the bones, generally near the articular ends (Fig. 171); these may soften and break down with abscesses, sinuses, etc., very much after the manner of a tuberculous inflammation (Fig. 172).

The lesions of the other long bones are essentially the same as of the tibia. They are nearly always symmetrical and often multiple. In



a case under my observation in a boy of four years, the disease involved both tibiæ, both radii, the right ulna, the left metatarsus, and the metacarpal bone of the left thumb. The course of syphilitic osteo-peri-



FIG. 172.—SYPHILITIC BONE LESIONS IN A BOY FOUR YEARS OLD. The lower end of the radius of both arms is enlarged as a result probably of former epiphysitis; there are sinuses leading to dead bone over the metacarpal bone of the right thumb, and over the upper extremity of the left ulna. The last two are recent lesions.

ostitis is very chronic, and some permanent deformity is the rule, unless cases come very early under treatment.

When affecting the bones of the cranium the disease usually takes the form of a gummatous periostitis, which leads to the formation of large nodes. These may remain as permanent deformities, or they may break down and suppurate, with necrosis of one or both tables of the skull. This may be followed by inflammation of the dura, the pia, and even of the brain itself.

**Symptoms.**—When the long bones are affected, the symptoms are pain, tenderness, and deformity. These come on very gradually, and often the deformity is noticed before either pain or tenderness is sufficiently marked to attract attention. The pain is regularly worse at night, and often felt only at that time; it may be mild and occasional, or so severe as virtually to prevent sleep. There is tenderness on pressure over the bones affected, the acuteness of which will depend upon the activity of the process. When suppuration occurs, it comes very slowly, and never with symptoms of acute inflammation. Sinuses usually continue to discharge until a sequestrum is exfoliated. The course

of the disease is very tedious, and the whole duration is usually several years.

When the cranium is affected, there are seen irregular nodes, especially upon the frontal and parietal bones. They are from one to two inches in diameter, and project from one-eighth to one-fourth of an inch above the general outline of the skull. There may be pain, tenderness, softening, suppuration, and necrosis, as in the long bones.

**Diagnosis.**—It is rare that disease of the bones of the cranium is due in childhood to any other cause than syphilis, and this disease may usually be assumed to exist if traumatism can be excluded. The bosses upon the cranium in rickets are always large, smooth, and regular in position, and belong to infancy.

Syphilitic disease of the long bones is recognised by the nocturnal pain, the tenderness and peculiar deformity, and by the association of other late manifestations of syphilis—i. e., the peculiar notched teeth, the interstitial keratitis, the enlarged epitrochlear glands, etc. Tuberculous disease generally affects the articular ends of the bones; syphilis nearly always the shaft. The diffuse hyperplasia of the tibia and the sabre-like deformity of its anterior border are rarely, if ever, due to any other cause than syphilis.

The deformities of the long bones have in some cases a certain resemblance to those due to rickets, but the two conditions can hardly be confused if a careful examination is made.

**Treatment.**—The constitutional treatment of these lesions is the same as that of the other late manifestations of syphilis; for details see the chapter on Syphilis. Surgical treatment is required in cases which terminate in necrosis, whether of the cranium or the extremities. They are to be managed like the same conditions in adults.

#### *Syphilitic Dactylitis.*

This belongs to a somewhat earlier period of syphilis than the disease just described, and is usually seen in infants. It is not a frequent manifestation of syphilis, and as compared with tuberculous dactylitis it is much less common and occurs at an earlier age. It was first fully described by Taylor (New York). The symptoms closely resemble the tuberculous form. It may begin as a periostitis, but more frequently as an osteo-myelitis. Like the tuberculous form, it may go on to suppuration and necrosis. According to Taylor, dactylitis is more often single than multiple, but in my own cases several phalanges have generally been involved, and the lesions have often been symmetrical. In one case, the first phalanx of every finger of both hands was affected. The Röntgen ray pictures show that the metacarpal bones are also involved in many cases (Figs. 175 and 176).

The symptoms and course of syphilitic dactylitis are essentially the

same as in the tuberculous form. The differential diagnosis is considered with the latter disease. The prognosis is much the same in the two varieties, with the exception that in the early stage the syphilitic cases may



FIG. 173.

FIG. 174.

**FIGS. 173, 174.—SYPHILITIC DACTYLITIS.** On right hand first phalanges of forefinger and little finger affected; on left hand first phalanx of thumb and second phalanx of second finger.

often be arrested by constitutional treatment. This is the same as in other late lesions of syphilis. The same local treatment should be employed as in the tuberculous cases.

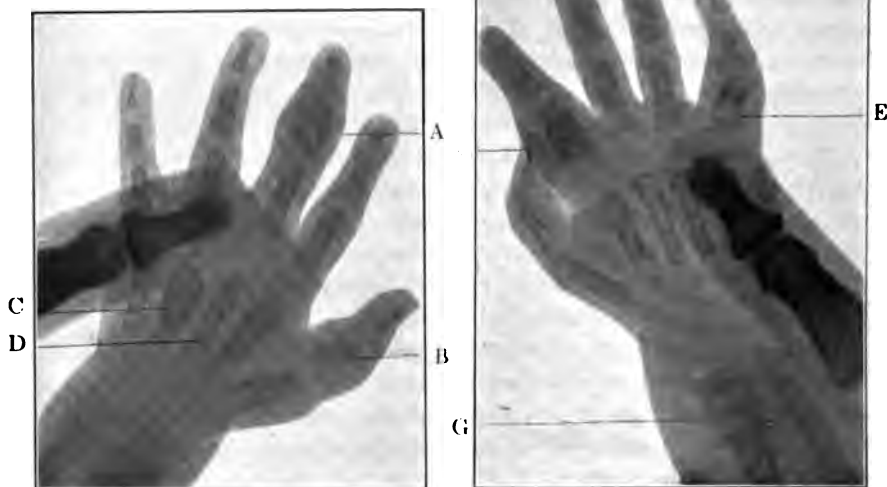


FIG. 175.

FIG. 176.

**FIGS. 175, 176.—RÖNTGEN RAY OF SAME HANDS.** Note that besides the bones shown in the other pictures, two metacarpal bones (*C, D*) are affected in the left hand and the lower end of the radius (*G*) in the right hand.

## CHAPTER V.

## DISEASES OF THE SKIN.

THE skin at birth is covered with a whitish sebaceous secretion, the vernix caseosa. The skin itself is of a deep purplish colour, which changes to a bright red over the face and trunk in a few minutes, with the establishment of normal respiration, and in a few hours the whole body has the same tint. This excessive redness slowly fades during the first month, at the end of which time the skin has assumed the pale pink of infancy. On the third or fourth day there may be seen the first signs of icterus; this generally fades by the end of the second week.

The epidermis which is present at birth soon loosens and is thrown off. This normal desquamation usually begins upon the fourth or fifth day, and is completed in ten days or two weeks. If the skin is frequently oiled and properly bathed, desquamation is scarcely noticeable unless a close examination is made. In some infants, especially those who are delicate and cachectic, it is very much more marked, and closely resembles that seen in scarlet fever. Ritter has described an *exfoliative dermatitis* of the newly born, appearing generally during the second and third weeks, a condition which is regarded by Kaposi as simply an exaggeration of the normal physiological desquamation. This process may be mistaken for that due to hereditary syphilis; the latter, however, is rarely general, appears later, and is much more prolonged.

Perspiration is rarely present before the end of the fourth month, and is then seen only upon the forehead. In healthy infants it is scarcely noticeable during the first year. Copious perspiration is most frequently a symptom of rickets; less marked perspiration may occur with any general weakness or during acute illness.

## CONGENITAL ICHTHYOSIS.

Congenital, or more properly fetal, ichthyosis, sometimes known also as diffuse keratoma, is a rare disease, characterised by the formation, usually all over the body, of a thick, horny epidermis resembling parchment. This is divided by fissures or shallow furrows into irregular patches; sometimes these are two or three inches wide, at others as small as a pin's head. The disease begins in the early months of fetal life, and is an abnormality in the development of the skin, there being an excessive proliferation of the layers of the epidermis.

**Symptoms.**—In the gravest form of the disease the child often lives but a few hours, and rarely more than a week. The openings of the nostrils and the ears may be occluded by the excessive production of epithelial cells. The eyes are in a condition of ectropion, and there are

often deformities of the mouth and other orifices due to the contractions of the skin. The nails and hair are usually imperfectly developed. The body seems incased in a hard, horny covering, and looks as if it had been varnished or covered with collodion. The skin cracks or splits and the edges curl up, an appearance which has been aptly compared to the skin of a boiled potato.

In the milder form, the duration of life is indefinite, depending upon the degree of development of the disease; but even in such cases there are frequently seen the deformities at the orifices of the body, and there may also be a continued exfoliation of the epidermis in large irregular



FIG. 177.—CONGENITAL ICHTHYOSIS IN A CHILD TEN MONTHS OLD. The large scaly patches are well shown on the lower part of the right chest and abdomen, and the constricting bands upon the legs. (From a photograph by Dr. Cabot.)

patches. After this has separated, the skin beneath appears red and moist, but gradually becomes dry, hard, and shining, slowly contracting until it splits in various directions. In a case recently under observation in the Babies' Hospital, a picture of which is shown in the accompanying illustration (Fig. 177), it was stated by the mother that during the first ten months of life complete exfoliation of the skin had occurred in the course of every two or three months.

The outlook is bad in all cases; in most of the severe forms death occurs in infancy, but in some of the milder ones, life may be prolonged throughout childhood. The "alligator boy" of the "Dime Museum" is an example of this class.

**Treatment.**—The indications are to keep the skin moist and soft by the use of oils, continuous baths, etc., and to prevent infection by perfect cleanliness. Although a certain amount of improvement usually follows these measures, a cure is not to be expected.

## MILIARIA.

The term miliaria is applied to an obstruction of the sweat glands, which may occur either with or without inflammation. The non-inflammatory form is known as *sudamina*, the inflammatory forms as *miliaria rubra*, *miliaria vesiculosa*, and *miliaria papulosa*.

**Sudamina.**—In this form there is no inflammation. The sweat ducts, according to Crocker, are blocked by an accumulation of epithelial cells while no perspiration is going on; and when the process is restored the fluid, being unable to escape, accumulates in the form of tiny vesicles. These appear like small pearly bodies very closely set, and disappear in the course of a few days by absorption. Fresh crops may appear from time to time. Sudamina may be seen in any of the continued fevers or exhausting diseases. It requires no treatment.

**Miliaria Rubra.**—This condition, also known as *red gum*, *strophulus*, etc., is a sweat rash, usually seen in young infants as the result of excessive clothing. It is most frequently observed upon the cheeks and neck, often upon the side of the face upon which the infant sleeps, or the side held against the mother's body while nursing, if this is done upon only one breast. The eruption consists of scattered red papules, sometimes with tiny vesicles. Miliaria rubra is an inflammation about the sweat glands, the result of which is a retention of their secretion. There is generally little or no itching. The treatment consists in the removal of the cause, and the application of some absorbent powder, such as boric acid and starch.

**Miliaria Papulosa** (*Lichen Tropicus*, *Prickly Heat*, etc.).—This is the most common and most important variety of miliaria. There is in this disease an obstruction of the sweat glands by inflammatory products. The lesion consists in the formation of bright-red papules, which are very closely set, the summits of some of them being surmounted by tiny vesicles, and here and there in severe cases even small pustules may be seen. If not interfered with by scratching, the vesicles dry up without rupture, and are followed by a slight desquamation. Where there is much scratching, an eczematous condition may result. Miliaria papulosa comes out with great rapidity, especially upon the neck, forehead, back, and chest. It is accompanied by an almost intolerable itching and stinging sensation. Over other parts of the body profuse perspiration occurs. The disease is produced by very hot weather and excessive clothing. Although the duration of a single attack is but two or three days, in

susceptible patients it may keep recurring for weeks, being exceedingly intractable. Where there is much scratching the resulting eczema is very troublesome. It is not infrequently followed by furunculosis.

The diagnosis of miliaria rubra and miliaria papulosa is usually easy. They are distinguished from eczema by the suddenness with which they appear, by the associated sweating of other parts of the body, by the transitory character of the eruption, and by the fact that the rash never occurs in circumscribed patches. Prickly heat sometimes resembles the rash of scarlet fever, but the fact that the tiny papules are in some places crowned by vesicles and that constitutional symptoms are absent, usually make the distinction an easy one.

*Treatment.*—Prickly heat is to be prevented by light clothing, frequent bathing, and the plentiful use of a good toilet powder, such as boric acid and starch. The skin should be protected against the irritation of flannel undergarments by the interposition of silk or linen. When the inflammation is at its height, relief is obtained by the application of a calamine and zinc lotion, or by a dilute solution of the acetate of lead; carbolic acid may be added to either, where the itching is intense. In some cases powders are preferable to lotions. One of the best is the stearate or the oxide of zinc, twelve parts; bismuth, three parts; powdered camphor, one part; or equal parts of starch and boric acid may be used, or simply rice flour. All of these must be very freely applied. The diet should be light and fluid, and if milk is the food it should be considerably diluted.

#### SEBORRHOEA.

Seborrhœa is considered by dermatologists generally, as a functional disease of the sebaceous glands; although Unna regards all such cases as inflammatory, and classes them as seborrhœic eczema, which is of parasitic origin. The disease may affect almost any part of the body, and children of any age, but the most frequent form is that which is seen upon the scalp in young infants. This is the most important variety, and the only one which will be here considered.

Seborrhœa of the scalp is characterised by the formation upon the vertex, of dirty-yellow crusts, which are soft, greasy, and friable. They are composed of epithelial cells, fat-globules, and granular masses, to which is always added dirt. In neglected cases the hairy scalp is nearly covered by a dense crust, which may be as thick as heavy pasteboard. If the crusts are removed the underlying scalp may be found perfectly healthy, but more frequently, in cases of long standing, it is eczematous. The eczema is set up by the decomposition of the exudation, or by the efforts to remove the crusts by such means as the fine-toothed comb, commonly employed in domestic practice. There is little tendency to spontaneous improvement or recovery, and the condition often lasts for

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months. Every seborrhœa should be treated, for when neglected it furnishes a favourable soil for the development of eczema.

**Treatment.**—Only local measures are required. The crusts are first to be softened with oil, and then removed by washing thoroughly with warm water and soap, after which an ointment of resorcin (resorcin, gr. x; ungt. aquæ rosæ, ʒj) or sulphur (precipitated sulphur, ʒj; lanoline, ʒj) should be applied. The oil and soap and water are repeated every few days, or as often as the crusts form. In the meantime the scalp is kept covered with the ointment.

#### ECZEMA.

Eczema may be defined as a catarrhal inflammation of the skin. It is the most frequent and altogether the most important disease of the skin in early life. The scope of the present work permits only a discussion of such features and varieties as are peculiar to infants and young children. The eczema of older children does not differ in any essential points from that of adults.

**Etiology.**—The conditions in infancy which predispose to eczema are, first, that the skin is extremely delicate, and hence more easily affected by external irritants and micro-organisms; secondly, its more intense glandular activity. While all children are susceptible, there are certain ones in whom the susceptibility is very marked, and in them the slightest amount of external irritation, or the most trivial disturbance of digestion may produce a severe eruption. We can not connect eczema with any single diathetic condition; but it is much more often seen in children with so-called gouty antecedents than in others. Such children are often in later life the subjects of asthma. Eczema of the face is common in fat, healthy-looking infants, both in those who are nursing and in those who are artificially fed. It rarely occurs in poorly nourished children.

The exciting causes of eczema may be external or internal. Of the former the most important are heat, cold dry air, and winds—as in the familiar chapping of the face—the use of hard water or of strong soaps in bathing. The disease may be due to the irritation of clothing, to want of cleanliness, or to irritating discharges from mucous surfaces, as in the eczema of the upper lip, thighs, or buttocks. It accompanies most of the parasitic skin diseases, particularly pediculosis, scabies, and ring-worm.

What part is played by micro-organisms in the etiology of eczema has not yet been fully determined. As a primary factor they do not seem to be of the first importance. Secondary infection, however, occurs in most cases, and this is important in keeping up the disease.

The internal causes of eczema are chiefly associated with deficient elimination from the kidneys and bowels, and digestive disturbances. It



often accompanies chronic constipation, especially when this is due to an excess of fat in the food. Eczema is also seen where the urine is scanty and concentrated because children partake too largely of solid food. The latter is true both in the first and second years.

Eczema may be produced by any form of digestive disturbance, but it is especially frequent in the intestinal indigestion which results from overfeeding, or the too early or excessive use of farinaceous food, or from breast-milk in which the percentage of fat is very high. Of farinaceous articles, the two which are most often to be blamed are potato and oatmeal. Although eczematous patients usually appear to be well nourished, it is rare that some symptoms of indigestion are not present.

Eczema is often due to some form of reflex irritation. Such are the cases which accompany dentition, and the rare ones due to genital irritation. By many writers the eczema caused by disorders of the stomach or intestines is regarded as reflex. The stronger the predisposition, the more trivial is the reflex irritation required to induce an eruption.

**Simple Chronic Eczema—Eczema Rubrum.**—This is the most frequent form of eczema occurring in infants and young children, and is usually seen upon the face. It affects by preference the cheeks, forehead, and scalp, not infrequently the ears and neck, and may occur upon any part of the body. Upon the trunk and extremities the eruption is usually in patches, but in rare cases may cover nearly the entire body. The disease generally begins upon the cheeks with the formation of small red papules; later these coalesce, and there is a moist, red surface exuding serum or sero-pus. The secretion dries and forms thick, gummy crusts, which may be so hard as to form a mask for the face. From the scratching caused by the almost intolerable itching, the surface bleeds freely, and the dried blood gives to the crusts a dirty-brown colour and adds to the distressing appearance. The skin is often much swollen. After the removal of the crusts there is seen, in acute cases, a red, inflamed, granular surface, discharging pus or serum and bleeding readily. When the process is less active, there is redness, thickening, induration, and scalliness of the skin, and marked itching. In the same case these stages may alternate, exacerbations occurring whenever the exciting cause is particularly active. From the cheeks the disease spreads to the forehead, ears, and scalp, and here similar lesions are seen. Upon the trunk and extremities thick crusts rarely form, but the skin is red, thick, and scaly. The parts most often affected are the forearms, chest, elbows, knees, abdomen, and back; occasionally the eruption is general. Eczema of the occipital region of the scalp is usually due to pediculosis.

Swelling of the lymph nodes in the neighbourhood of the eruption is a constant feature of eczema of the face and scalp; these may reach the size of a chestnut or walnut, and occasionally they may suppurate. Intense itching is a characteristic feature of all cases of eczema of the face.

or scalp. While most children with eczema are well nourished in the beginning, and some remain so during a prolonged attack, the general health of many is undermined. The itching and discomfort cause constant irritability, loss of sleep and other nervous symptoms which sometimes seriously impair the child's nutrition.

The effects of very extensive eczema resemble in some particulars those of burns of the second degree. There may be fever, delirium, other nervous symptoms and even a fatal termination. Four cases have been seen recently in the Babies' Hospital with a generalised eczema in which there developed, without evident cause, exceedingly high temperature, in two cases reaching 109° F., accompanied by symptoms of a most profound intoxication. Two of the four cases ended fatally; one child in whom the temperature reached 109° F. recovered. In two of the patients a marked degree of acetonuria was present. No satisfactory explanation of these severe intoxications has yet been offered.

There are some patients in whom an alternation of eczema and attacks of bronchitis with asthma may occur. During the eczema, the pulmonary symptoms are entirely wanting; but when the eczema is relieved the pulmonary symptoms rapidly develop. This is often seen in patients of the so-called gouty diathesis. In a few patients an alternation of eczema and diarrhoea is observed.

Eczema of the face is very chronic, easily improved, but cured only with great difficulty. There is a strong tendency to relapse, brought on by neglect of local treatment or by any digestive disturbance.

The predisposition to eczema often ceases with the second year; those who have suffered from it almost constantly during infancy may be free from it during the remainder of childhood. This is in part to be explained by the loss of fat in consequence of more active exercise and a diet which is more largely nitrogenous. Where the disease continues through the third and fourth years, the associated infantile condition—obesity—is not infrequently present.

**Pustular Eczema of the Scalp.**—This condition, often called "simple impetigo," is less frequently seen in infants than in children from two to five years old. There are usually present from half a dozen to fifty greenish-yellow crusts, matting the hair, usually discrete, but sometimes coalescing to form a mask over half the scalp. There is very little itching, in some cases none at all. The lymph glands are invariably enlarged. There is frequently continued auto-infection, and in this way the disease may be prolonged indefinitely. It is possible, too, that infection may spread to other children.

**Intertrigo.**—This term is rather indiscriminately applied to any eruption which develops upon two moist surfaces, which are in contact. It is often regarded as a form of eczema. There may be a simple erythema or an eczema resulting from traumatism or the decomposition of secre-

tions. Intertrigo is seen in the folds of the groin, between the scrotum and the thighs, between the buttocks, about the anus, in the axillæ, in the neck, or behind the ears. Its essential causes are moisture, friction, want of cleanliness, and sometimes infection. The disease is generally seen in its worst form about the thighs, genitals, and buttocks; it sometimes covers the sacrum and extends down to the middle of the thighs. There is an intense uniform redness, and in some cases the epidermis is denuded over large areas, and the surface is moist. There is no thick crusting and little or no itching. Intertrigo is usually easy to control except in very poorly nourished or marantic children, among whom it is especially frequent.

**Diagnosis of Eczema.**—This is usually quite an easy matter. In the majority of cases, the disease affects the face or the scalp, and its appearances are typical. Eczema of the body or extremities may be confounded with scabies or syphilis, and occasionally with other forms of skin disease. Scabies resembles eczema in its intense itching and multiform lesions; but in the former, one may often find evidences of its presence in other members of the family; the parts most frequently affected are the flexures of the wrists, the elbows, the skin between the fingers, the margins of the axillæ, the lower part of the abdomen and back, and, in boys, the penis; and by careful examination with a lens some of the characteristic burrows are certain to be discovered.

Syphilis is likely to be confounded with papular eczema of the buttocks. The latter affects the parts near the anus, and the irritation may lead to the development of spots closely resembling mucous patches. The local appearances may at times be indistinguishable from syphilis, and the diagnosis is to be made only by the other symptoms present. In syphilis the characteristic eruption is seen usually upon the face, hands, legs, and sometimes the palms and soles; there is no itching and very little evidence of inflammation; the eruption is copper-coloured, and occurs as small circumscribed spots; there are usually present other symptoms, such as the coryza, the syphilitic cachexia, and enlargement of the spleen.

The diagnosis from pediculosis and ring-worm of the scalp, rarely presents any difficulties.

**Prognosis.**—All cases of chronic eczema are tedious. There is only a slight tendency to spontaneous improvement, and very little to spontaneous recovery during early infancy. About the end of the first year the disease disappears in many children; some relapse after this time, but others are never again troubled with eczema. In a severe case of general eczema the possibility of the development of severe toxic symptoms should not be forgotten. In a given case, the prognosis depends upon the duration of the disease, its severity, and very much upon the co-operation of the mother or nurse. The results obtained depend not

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only upon the particular line of treatment adopted, but upon how well it is carried out. Usually it must be continued for several months. Eczema of the face is especially intractable when occurring in children suffering from chronic indigestion and constipation. Intertrigo is in most cases easily cured, unless the patient is suffering from marasmus.

**Treatment.**—A judicious combination of general and local measures is necessary for the best results. One should first seek to discover and correct what is wrong with the child's digestion, assimilation, and elimination; unless nutritive disturbances can be removed, local treatment will give only temporary relief. External causes also must be investigated. The local measures employed must be chosen with reference to the condition present; stimulating applications should not be ordered for an acutely inflamed skin, nor sedative applications in very chronic conditions.

**Diet.**—A thorough investigation into the food is necessary, not only as to its character, but as to quantity and preparation, the manner and frequency of feeding, etc. If the patient is a nursing infant, an examination of the nurse's milk is indispensable to intelligent treatment. If the child is very fat and well nourished, it is generally the case that the fat of the milk is too high and must be reduced according to the rules given elsewhere, the most important thing being to exclude from the nurse's diet malt liquors and alcohol in all forms, and reduce the amount of meat. The amount of food should be reduced by lengthening the period between the nursings, and shortening the time which the child is allowed to remain at the breast at one nursing. Plain water, or better, some alkaline water, should be given freely between the nursings. In children fed upon cow's milk, the trouble may be with the sugar, or more frequently with the fat. This should first be reduced and if no improvement occurs the sugar should also be diminished. In all dietetic changes the general nutrition of the child should be regarded as more important than the relief of the eczema.

During the latter part of the first and the entire second year, the usual error is that of overfeeding with in most cases an excessive use of solid food, especially farinaceous articles. The diet should then be much reduced, and the amount of farinaceous food restricted, potatoes and oatmeal being absolutely prohibited. The diet which suits most children best is one composed of milk, beef juice, broth, fruit, eggs, and a little red meat, with the addition in some cases of rice, wheat, or barley. In severe and obstinate cases, however, as complete a change in diet as possible is sometimes the best prescription. Any form of indigestion which exists is to be managed according to the special indications in each case.

The diet of older children needs to be watched no less closely than that of infants. The general rules laid down elsewhere for feeding after

the second year should be observed. The great majority of cases do best upon a diet which is largely fluid, and composed of milk, buttermilk, kumyss or matzoon.

Elimination by the kidneys should be stimulated by the very free use of water, to which may be added the citrate, or acetate of potassium, from ten to twenty grains daily.

Attention to the condition of the bowels is of the greatest importance. To overcome the tendency to constipation is in many cases to cure the eczema. Suggestions under this head will be found in the chapter on Chronic Constipation. The occasional use of catharsis by calomel every week or ten days is often beneficial. The best effects from this are seen in overfed children. It has a favourable effect upon the kidneys as well as upon the bowels. The bowels must not only be opened, they must be kept open by the daily use, if necessary, of some of the milder laxatives, such as magnesia, phosphate of sodium, rhubarb, or cascara. Castor oil, given in from half a teaspoonful to teaspoonful doses every night for two or three weeks at a time, is at times a useful measure.

When the disease occurs in flabby, anæmic, or poorly nourished children, iron and bitter tonics are required, but rarely cod-liver oil. In other words, the child's general condition should be treated just as if no eczema existed. Arsenic is indicated in a chronic or recurring form of eczema with dry, scaly eruption. It is in no sense a specific remedy, but is sometimes of value.

The *general management* of cases is important. The skin must be carefully protected by an ointment whenever the child is in the open air; if the weather is very cold, or there are high winds, children with active eczema should not go out, but be aired indoors. Never should an eczematous surface be washed with plain water, and much less with castile soap and water, so frequently employed by the ignorant. Where washing is necessary, it may be done with bran water, milk and water, or starch and water, to which borax (a teaspoonful to the quart) may be added. The clothing should not be so excessive as to keep the child constantly in a perspiration. Napkins should not be washed in strong soda solutions, nor, in case of eczema of the buttocks, should they ever be used a second time after being simply dried.

In eczema of the face it is absolutely necessary to prevent the child from scratching the parts. The use of a mask is not always sufficient, nor the wearing of mittens; nor is the local application of anti-pruritic lotions or ointments invariably successful. In severe cases mechanical restraint is absolutely indispensable. The most satisfactory method is to surround the arms at the elbows by pasteboard splints, and hold them in place by bandages. This allows free use of the hands, but makes it absolutely impossible for the child to reach the face.

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*Local Treatment.*—Local treatment is always necessary, for not only are the causes sometimes entirely external, but the condition may persist after the original internal cause has been removed. There are several indications to be met by local treatment at different stages in the disease: (1) To remove crusts and other inflammatory products; (2) to allay congestion and acute inflammation; (3) to relieve itching; (4) to protect the delicate new skin which is forming; (5) to prevent infection; (6) to stimulate the skin in the chronic stages of the disease.

Preparatory to the use of any application, the scales, crusts, and other products of inflammation must be softened and removed in order that the diseased surface may be reached. In most cases it is sufficient to soften the crusts by the use of olive oil for twelve or twenty-four hours, and then remove them by soap and warm water. If the crusts are very hard and thick, they can be softened by a poultice. During the stage of acute inflammation only sedative applications should be used. One of the best of these is a lotion of zinc and calamine:

|                                  |         |
|----------------------------------|---------|
| ℞ Pulv. calaminæ preparatæ ..... | 3 ij    |
| Zinci oxidi .....                | 3 ss.   |
| Glycerinæ .....                  | 3 j     |
| Liquor calcis .....              | 3 ij    |
| Aquæ rosæ .....                  | 3 viij. |

A piece of muslin should be dipped in this solution, and applied to the affected part, being kept in place by a bandage. If there is much itching, one per cent of carbolic acid may be added.

Another plan of treatment, where there is much secretion, is to keep the surface covered with equal parts of boric acid and starch or talcum powder. An application which is often successful in allaying the intense burning and itching is black wash. This is applied several times a day in full strength or diluted and allowed to dry on, after which a protective ointment is used.

A soothing application in general eczema is one composed of equal parts of lime-water and sweet-almond oil; sometimes this may be advantageously followed by smearing the body with a thick starch paste and allowing it to dry on.

As a simple protective ointment, one containing starch, zinc oxide, or bismuth, either alone or in combination, may be used. An excellent formula is Lassar's paste:

|                          |       |
|--------------------------|-------|
| ℞ Acidi salicylici ..... | gr. x |
| Zinci oxidi .....        | 5 ij  |
| Amyli .....              | 3 ij  |
| Vaseline .....           | 3 j.  |

Later, when the inflammation is less acute and the itching severe, nothing is so generally useful as a combination of tar and zinc, as in the following:

|                                    |        |
|------------------------------------|--------|
| <b>R</b> Ungt. picis liquidæ ..... | 3 iij  |
| Zinci oxidi .....                  | 3 iss. |
| Ungt. aquæ rosæ .....              | 3 vi.  |

For more chronic cases, the amount of tar may be increased. All ointments used should be spread upon muslin, and kept in close contact with the inflamed part by means of a bandage or mask. Little or nothing is accomplished by simply rubbing the ointment upon the affected part. Where it is difficult to keep a mask applied, or in situations where it is impossible to use the ointment, Pick's paste may be tried :

|                                  |        |
|----------------------------------|--------|
| <b>R</b> Pulv. tragacanthæ ..... | 3 j    |
| Glycerinæ .....                  | 3 iss. |
| Aquæ rosæ .....                  | 3 iv.  |

To this may be added zinc oxide, gr. xl, and carbolic acid, gr. v, or tar, ℥ x. A similar basis for ointments, made from gum tragacanth has been suggested by Elliot and is known as bassorin paste. It may be combined with tar, zinc, salicylic acid, or resorcin. An ointment containing five or ten per cent of calomel is often the best application for an eczema which is not too extensive.

The methods of treatment above mentioned are especially applicable to eczema of the face and scalp. For pustular eczema of the scalp the best application is the white precipitate ointment, which should be combined with three or four parts of vaseline. This is excellent also for small eczematous patches upon the body, but it is not to be used over a large surface.

In intertrigo, the treatment should have reference to the pathological condition which is present. Cases of simple erythema usually yield promptly to cleanliness and the free use of absorbent antiseptic powders, such as boric acid and starch in equal parts, or calomel two per cent may be used with talcum. If there is an acute dermatitis, the calamine and zinc lotion may be used, and later some protecting ointment. When infection has been added, lotions of resorcin or ichthyol, one-half or one per cent strength, should first be applied, and the skin then covered with one of the powders mentioned; both are to be repeated as often as the parts are wet by urine or soiled by fæces. It is important in all cases that the diseased surfaces should be kept separated, which is best done by boric acid and starch. All napkins should be immediately removed when soiled.

In cases of chronic eczema, where the skin remains thickened, red, scaly, and itching, stimulating applications are to be used, such as the tincture of green soap or stronger preparations of tar than those mentioned. They should be applied every three or four days.

## FURUNCULOSIS.

A furuncle, or boil, is a circumscribed inflammation of the subcutaneous cellular tissue, usually beginning in a hair follicle, and usually ending in suppuration. When severe, it may result in necrosis of the follicle, which forms the "core," or the necrotic process may extend to the surrounding tissues for a variable distance. The ordinary boil need not be described, as it presents nothing peculiar in early life. The condition, however, which is characteristic of young children is the formation of small ones in great numbers. It is to this more especially that the term *furunculosis* is applied. The principal location of these small abscesses is, in nearly all cases, the scalp, face, and shoulders, although they may be found upon any part of the body. They are sometimes numbered by hundreds, and appear in crops for a period of several months. In size, they usually vary from a pea to an almond, and they rarely contain a core. Infants are much more often the subjects of this disease than are those who have passed the second year. In the great majority of cases the condition is not serious, yet, occurring, as it often does, in infants who are already suffering from extreme malnutrition or marasmus, whose tissues possess but little resistance, the process may develop into a gangrenous dermatitis, which may prove fatal.

Furunculosis is seen in children who are in other respects apparently healthy, even robust; but the majority are in a more or less debilitated condition, and often are the subjects of digestive disturbances. The disease is quite frequent in syphilitic infants; but these simple abscesses are to be sharply distinguished from those which result from the breaking down of gummata of the skin. Want of cleanliness of the skin is a factor of some importance in producing the disease. Furunculosis may be associated with eczema. The exciting cause in all cases, as shown by recent investigations, is the entrance of the *staphylococcus pyogenes aureus*, sometimes with other organisms, into the follicles of the skin.

**Treatment.**—The internal treatment is to be directed toward any disturbance of digestion or general nutrition which is present. General tonics are indicated in most cases, particularly iron, arsenic, and the compound syrup of the hypophosphites. But little reliance can be placed upon drugs such as sulphide of calcium, for the purpose of arresting this disease. Striking benefit, however, often follows the internal use of yeast; either brewer's yeast or the ordinary commercial yeast cake, freshly made, may be given. The latter is usually easier to obtain. To a child of two or three years one-fourth to one-half a yeast cake or about half a teaspoonful may be administered daily. Local treatment should have for its first object thorough cleanliness of the skin. This is best secured by frequently bathing the parts affected with a 1 to 5,000 solution of bichloride. Single furuncles may often be aborted by touching them



with pure carbolic acid or the application of Bier's cups. In my experience the best plan of treating the multiple small furuncles, is to delay incision until they have pointed, then to incise and empty the follicle completely by compression. After this the part should be covered with simple collodion. Where the abscesses are of large size and upon the scalp, it is wise to make compression by applying a snug bandage for a day. When the suppuration is more diffuse and there is necrosis of the cellular tissue, ichthyol, either in the form of an ointment or lotion (one to five per cent strength) is one of the best applications. For general furunculosis or the continual recurrence of larger abscesses the use of vaccines is altogether the most effective treatment. While autogenous vaccines are perhaps preferable, the use of stock vaccines is in most cases equally effective. Injections should be repeated every three or four days; beginning with fifty millions, the dose may be increased to one hundred millions. The results in most cases are very striking.

#### GANGRENOUS DERMATITIS.

This is not a frequent disease, and is seen almost exclusively in infancy. It may be primary or it may follow other diseases, and hence has been described under many different names, viz., *varicella gangrenosa*, *ecthyma gangrenosa*, *pemphigus gangrenosa*, etc.

The lesion consists in small, discrete areas of inflammation of the skin, ending in necrosis. In the primary cases there is usually first seen a vesicle, about as large as a pea, with a dusky areola; it increases in size and becomes a pustule. Crusts form which are quite adherent, and on removing them a loss of tissue is seen. The ulcers usually have sharp but not undermined edges, often presenting a "punched-out" appearance. By the coalescence of several small ones, ulcers an inch or more in diameter are sometimes formed.

The primary form of gangrenous dermatitis occurs in wretched, poorly-nourished infants, and is most often seen upon the buttocks. In this location it may be mistaken for syphilis. The secondary form is more common, and usually follows varicella, less frequently vaccinia, or impetigo. In such cases the lesion is usually seen upon the upper half of the body, especially upon the neck and chest. It follows the ordinary lesions of varicella and continues usually, in spite of treatment, from one to four weeks, in many cases ending fatally. The disease always occurs in infants of poor vitality, often in those suffering from marasmus, and is seldom seen outside of institutions. It may be accompanied by fever, and other severe constitutional symptoms.

For the production of the disease, two factors are necessary: First, the constitutional condition referred to; and, secondly, the entrance of pyogenic germs, usually the streptococcus pyogenes.

**Treatment.**—Every means possible should be employed to build up the general health of the infant by tonics, fresh air, careful feeding, etc. Locally, strict cleanliness and antiseptic applications are necessary. The best application is a solution of bichloride (1 to 5,000), or an ointment of ichthyol or white precipitate.

#### IMPETIGO CONTAGIOSA.

Impetigo contagiosa is a disease characterised by the formation of discrete vesiculo-pustules, occurring most frequently upon the hands and face. Cases are usually seen in groups affecting several children in one family or institution. It may be communicated from one person to another, and spread by auto-inoculation from one part of the body to another.

One rarely has an opportunity to see the disease until vesicles have formed. These are usually from one-fourth to one-half inch in diameter, and are flaccid, never distended. Later, their contents become slightly yellowish; then they rupture and dry, forming thick yellow crusts, which have the appearance of being "stuck on," the surrounding skin being quite healthy. After the crusts fall off, a small red patch remains, which slowly fades. The true skin is not involved, except in poorly nourished, cachectic subjects, as a result of continued local irritation, like scratching. Under such conditions ulceration may occur. Instead of the small vesiculo-pustules described, bullæ from one to two inches in diameter may form, filled first with serum, afterward with sero-pus. Very little inflammation is seen about these patches, and in most cases the intervening skin is normal.

The favourite seat of the eruption is the face, especially about the chin, next the hands, the neck, the feet and legs, the forearms, and the scalp; it is rarely seen upon the abdomen, and never upon the back. There may be only half a dozen vesiculo-pustules, or from thirty to forty may be present. The smaller ones sometimes coalesce and form others of considerable size. Itching is never a prominent symptom, and in most cases it is absent altogether.

The usual duration of impetigo contagiosa is two or three weeks; it, however, runs no regular course, and by continued auto-inoculation may last much longer than this.

The studies of Gilchrist (Baltimore) point to a streptococcus of low virulence as the cause of this disease. European investigators, however, have with considerable uniformity found the staphylococcus pyogenes aureus in the vesicles. Impetigo contagiosa may occur in any child, but is seen most frequently in one who is poorly nourished.

The diagnosis is not often difficult, and is made by the following

features, viz., the occurrence of several cases together, the isolated vesiculo-pustules situated upon the face and hands, the slight itching, and the prompt cure by local measures only. The bullous form, however, is frequently confounded with pemphigus; many cases in which the diagnosis of pemphigus is made are examples of impetigo.

**Treatment.**—This is simple and usually very effective. The crusts are to be softened and removed by thoroughly washing the part with soap and water or a bichloride solution, after which the white precipitate ointment, combined with three parts of vaseline, should be applied.

#### URTICARIA.

Urticaria is a frequent disease in early life, and presents some features, particularly in infants and young children, which are quite different from those seen in adults. This is due to the fact that papules and vesicles, and occasionally pustules, are associated with the wheals. As the wheals quickly subside, it frequently happens that the other lesions mentioned are the only ones present. This fact has given rise to considerable confusion in names, and the urticaria of infancy has been called *lichen urticatus*, *urticaria papulosa*, *strophulus*, etc. It is now pretty generally agreed that the clinical picture, which is a familiar one, belongs to a single disease, and that this is urticaria.

The initial lesion is the wheal, but on account of the extreme susceptibility of the skin in young children, the process is more intense than in older patients, so that it may result in the formation of an inflammatory papule or a vesicle. In a few hours the wheals may subside, and only the papules or vesicles remain, and without a good history the disease may be a very obscure one. The papules and vesicles occur with greatest frequency upon the hands and feet, particularly the palms and soles. The more severe form of the disease in poorly nourished children is sometimes accompanied by a pustular eruption, and there may even be deep ulceration (ecthyma). The usual appearance of the eruption is a number of small inflamed red papules whose tops are covered with crusts, the result of scratching. The eruption may be limited to the extremities or it may be general. It is as a rule more severe in regions accessible to scratching.

There is usually severe itching, which leads to loss of sleep, and often in this way the disease affects the general health of the child. The urticaria of older children does not differ essentially from the same disease in adults. The alternation of urticaria and asthma is occasionally met with.

The character of the eruption in urticaria and even its distribution strongly suggest scabies; and unless one has had an opportunity to witness the development of the lesions, a differential diagnosis may be very difficult,

as almost every lesion, except the wheal, may be identical in both diseases. Other cases may resemble varicella.

Urticaria in early life is most frequently the result of some disturbance in the digestive tract. Almost any sort of derangement may produce it, the exciting cause varying with the patient. Exceptionally, it may result from other forms of irritation, such as dentition or intestinal worms.

**Treatment.**—The milder forms of urticaria usually respond quickly to treatment; but when it is severe and has existed for several weeks, it is one of the most troublesome and intractable skin diseases of childhood. The treatment is to be directed primarily toward the condition of the digestive organs. Children should be put upon a milk diet. The bowels should be kept freely open by calomel, a nightly dose of castor oil, or a morning dose of magnesia. If the urine is excessively acid and scanty, alkaline diuretics should be given. The drugs most useful for the indigestion with which urticaria is associated are salicylate of soda and nitro-muriatic acid, each of which is to be given after meals.

All local causes of irritation, such as rough flannel underclothing, should be removed. The sleep may be so much disturbed as to require the use of trional or bromide and chloral. Antipyrine and atropine are often useful; they may be given separately or in combination, and in moderately large doses.

The local irritation and itching may be relieved by a very dilute solution of the subacetate of lead or carbolic acid, or by a mixture of vinegar, or the fluid extract of hamamelis, or bicarbonate of soda, and water. When pustules are present, the white precipitate ointment may be used, combined with four parts of vaseline; in the papular and vesicular forms, an ointment of ichthyol, one per cent strength. In many cases the improvement in the general health by the use of tonics, change of air, etc., will accomplish more than any measures directed especially to the relief of the urticaria.

#### SCABIES.

Scabies is a contagious disease due to the burrowing into the skin of the female acarus, with secondary lesions which result from scratching.

The burrowing of the acarus is usually where the skin is thinnest—viz., between the fingers, on the flexor surfaces of the wrist, the axillæ, and, in males, the genitals. It is not seen upon the face, except in infancy, when it may be infected by contact with the breasts of the mother. The lesion excited by the acarus is usually a papule or a vesicle, sometimes a pustule. In some cases no evidences of inflammation are present, but in infants and young children they may be marked—pustular eruptions being frequent and often extensive, especially upon the hands

and feet. The characteristic burrow is from one-fourth to one-half inch in length, and appears as a fine brown or black line, at the end of which the acarus may be discovered as a small white speck. The burrows are often difficult to find in infants. They are generally to be seen along the ulnar border of the hand and between the fingers. The intensity of the inflammatory lesions varies greatly in different cases; in some they are very few, while in others, particularly in delicate, cachectic, and neglected children, they are sometimes very severe, so that the skin of the affected part is nearly covered with pustules. These secondary lesions are due to infection by the streptococcus or staphylococcus. A pustular eruption upon the hands should always suggest scabies. The lesions which result from scratching may be found on any accessible portion of the body. There are usually at first linear, bloody marks, but after a time these may not be visible. In little children urticaria is often associated.

The diagnosis of scabies is usually quite easy, as several children in a family are likely to be affected, particularly if they occupy the same bed. The diagnostic features of the eruption are the presence of papules, vesicles, or pustules, especially upon the hands, wrists, and genitals. A careful examination with a lens will usually disclose some of the characteristic burrows, or even the acarus. In infancy, scabies may be easily confounded with the vesicular form of urticaria, unless the development of the lesions has been observed.

Scabies may always be cured, provided sufficient precautions are taken to prevent reinfection. This necessitates boiling or baking, not only the patient's clothes, but all the bedding as well.

**Treatment.**—This should always be begun by a hot bath, in order to soften the epithelial scales about the burrows. The body should be thoroughly scrubbed with soap and water, preferably with a nail-brush, the bath being continued for at least half an hour. It is well to do this at night. After the bath, the body is anointed with the parasiticide, which should be thoroughly rubbed into the skin, clean clothing applied, and the child put into a perfectly clean bed. In the morning the ointment may be washed off, but none of the clothing previously worn should be put on. This treatment is to be repeated on two or three successive nights, and if thoroughly done it will effect a cure. The ordinary sulphur ointment is too irritating for use in little children, and one of the following may be substituted: Naphthol, 15 parts; creta preparata, 10 parts; vaseline, 100 parts (Kaposi); or, precipitated sulphur, 1 part; balsam of Peru, 1 part; vaseline, 8 parts; or the simple balsam of Peru may be applied without dilution. After the use of the parasiticide there is generally required, for a few days, some soothing application like those mentioned in the chapter upon Eczema.

## TINEA TONSURANS—RING-WORM OF THE SCALP.

Ring-worm of the scalp is a very frequent disease in institutions for children, often occurring as an epidemic. According to Crocker, the primary lesion consists in a red papule surrounding a hair, which soon increases to a small circular patch; this spreads at its outer margin, gradually increasing in size until it is from one to two inches in diameter, but rarely larger than this. Sometimes several of the patches coalesce. These affected areas always have rounded borders, and are sharply outlined. Here the hairs are very brittle, and often broken off close to the scalp, so that the area may appear to be bald. Where they have not fallen off, the hairs have lost their lustre. The stumps of the broken hairs point in all directions.

The fungus which produces the disease is the *trichophyton tonsurans*. It penetrates the shaft of the hair, both the spores and the mycelium being seen under the microscope. The spores are present in great numbers in the hair, but the mycelium is most abundant in the scales. The amount of inflammation found in the diseased areas varies much in the different cases. There may be only a scaliness of the scalp, or a formation of pustules in the hair follicles, the hairs loosening and falling out in consequence. In young infants, where the hair is scanty and thin, the disease resembles *tinea circinata*—i. e., it is superficial, and the hair follicles are often not involved. Children of all ages are liable to *tinea tonsurans*. It flourishes particularly in institutions and in those children who are dirty and poorly cared for.

The diagnostic feature of the disease is the presence of scaly patches, with loss of hair. The patches are usually circular, and by examination with a lens the stumps of broken hairs are seen all over the diseased area. By a microscopical examination the fungus is discovered. In typical cases the diagnosis is easy if the process is at all advanced, but there are many atypical forms and many mild cases where the recognition of the disease is difficult. The symptoms are often masked by the inflammatory conditions present. The disease may be confounded with *seborrhœa*; but in the latter the lesion is diffuse, never sharply defined; there is general thinning of hair over the scalp, and never the stumpy, broken hairs. *Psoriasis* has points of resemblance, but it is usually found on other parts of the body, especially the knees and elbows, and upon the scalp the patches are more numerous and smaller. In *eczema* the loss of hair in circumscribed patches is never seen, nor are the broken stumps.

*Tinea tonsurans* is always curable, provided the patient can be kept under close surveillance, and treatment thoroughly carried out. There is no tendency to spontaneous recovery. In a recent case, treatment must usually be continued for one or two months, and in chronic cases from six months to one year, with the closest watchfulness.

**Treatment.**—The great difficulty in treatment is to get the parasiticide deeply enough into the scalp to reach the fungus, since this is often at the very bottom of the hair follicles. As a first step, the hair should be cut short all over the patch and for at least an inch beyond it; this is necessary in order to get at the diseased part and to detect new foci of infection early—if possible before the fungus has extended deeply into the follicles. The parasiticide should be applied not only upon but around the patch, and the entire scalp should be washed thoroughly two or three times a week. To prevent the disease spreading, all the scales are to be kept softened by the use of carbolic soap. The hair should not be brushed, as this tends to scatter the spores and spread the disease. All patients, while under treatment, should wear a cap of muslin or oiled silk, or one lined with paper, in order to prevent infecting others. In institutions, affected children should invariably be isolated.

To destroy the fungus almost every germicide on the list has been advocated at one time or another, which proves that the disease is a very obstinate one, and that no one application is invariably successful. Those which have the sanction of the widest use are the tincture of iodine, the bichloride, white precipitate, and oleate of mercury, kerosene, creosote, and croton oil. As a vehicle for ointments, *adeps lanæ* (lanoline) is greatly to be preferred to vaseline or lard. Most of the germicides mentioned are used in the strength of one to five per cent, according to the age of the child and the irritability of the scalp. In an epidemic of ring-worm in the New York Infant Asylum the following combination of bichloride and kerosene proved extremely satisfactory: ten grains of the bichloride were dissolved in alcohol, and to this were added two and a half ounces each of olive oil and kerosene.

Epilation is necessary in many cases as an accessory to the application of germicides, particularly in older children.

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## CHAPTER VI.

### *DISEASES OF THE EAR.*

#### ACUTE OTITIS.

OTITIS is a frequent affection during infancy and early childhood, attacks usually occurring in the cold season. Of all the inflammatory conditions which may be met with in early life, there is perhaps none which more frequently gives rise to obscure febrile symptoms than this.

**Etiology.**—Acute otitis is, as a rule, a secondary disease, and is generally preceded by some infectious process in the rhino-pharynx. The usual avenue of infection is through the Eustachian tube.

The most severe forms of otitis usually follow scarlet fever, epidemic

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influenza, measles, diphtheria, or pneumonia. The entrance of fluids through the Eustachian tube from the nasal douche or nasal syringing may cause acute otitis. It sometimes results as an extension of inflammation from meningitis, especially the cerebro-spinal form.

The micro-organisms concerned in the production of acute otitis vary somewhat with the condition of which it is a complication. In the order of frequency there are found the staphylococcus aureus, the pneumococcus, the streptococcus, and the influenza bacillus. Mixed infections are very common. In cases complicating diphtheria, the Klebs-Loeffler bacillus may be found with any of the forms mentioned, or may occur alone. In chronic cases any of the pyogenic organisms may be present, and not very infrequently the tubercle bacillus.

**Lesions.**—The ordinary course of events in the pathological process is, first, acute hyperæmia and swelling of the mucous membrane of the rhinopharynx, which extends into the Eustachian tube, causing obstruction more or less complete. The inflammatory process may be limited to the tube, or it may extend to the mucous membrane lining the middle ear.

There are two varieties of acute inflammation of the middle ear: (1) The catarrhal form, which usually accompanies simple catarrh of the rhinopharynx or complicates measles. This is an inflammation of the mucous membrane merely, and its products are serum and mucus or muco-pus. It is not usually accompanied by great pain or followed by serious consequences. It is generally confined to the lower part of the tympanic cavity, and is the form most frequently seen in infants. (2) The purulent or phlegmonous form, which affects older children principally. This is a much more serious inflammation, and is often excited by the infectious catarrh of scarlet fever, diphtheria, or epidemic influenza. In this variety micro-organisms find their way into the middle ear in great numbers, and set up an inflammation of a more or less virulent type, which may involve not only the mucous membrane lining the tympanum, but also the cellular tissue in the upper part of the tympanic cavity. The lining membrane of the mastoid cells is involved in many, if not all, of the cases.

The catarrhal form of inflammation frequently subsides in a few days with proper treatment, the only result being a slight deafness, which is temporary. The phlegmonous form causes a stoppage of the Eustachian tube, rupture or sloughing of the tympanic membrane, and discharge of the products of inflammation, or rarely pus finds an outlet by burrowing between the cartilages. The inflammatory process may extend to the bones, causing necrosis of the ossicles or the bony walls of the tympanum. The remote results are periostitis and necrosis of the petrous bone, pachymeningitis, infectious thrombosis of the lateral sinus, general purulent meningitis, and cerebral abscess. These will be considered under Complications.



**Symptoms.**—These are usually few in number, but present great variability as regards their combination and intensity. The two most constant symptoms are pain and fever. In a typical case in an infant, there is generally at the beginning some discharge from the nose, slight congestion of the pharynx and tonsils, and a temperature of 100° to 102° F. There is nothing characteristic about this catarrh. After two or three days the objective symptoms subside, but the infant continues to be restless, worries much of the time, wakes frequently at night with a start, nurses poorly, and the temperature remains elevated, usually from 100° to 103° F. (Fig. 178). The infant seems decidedly ill, and yet no very definite symptoms are present. Sometimes there is marked tenderness about the ear, and the child refuses to lie upon the affected side, or shows signs of pain when the ear is touched. After a week or ten days spontaneous rupture of the drum membrane takes place, and subsidence of the constitutional symptoms follows. In some cases there is seen only a high temperature, ranging from 101° to 104° F., which persists for several

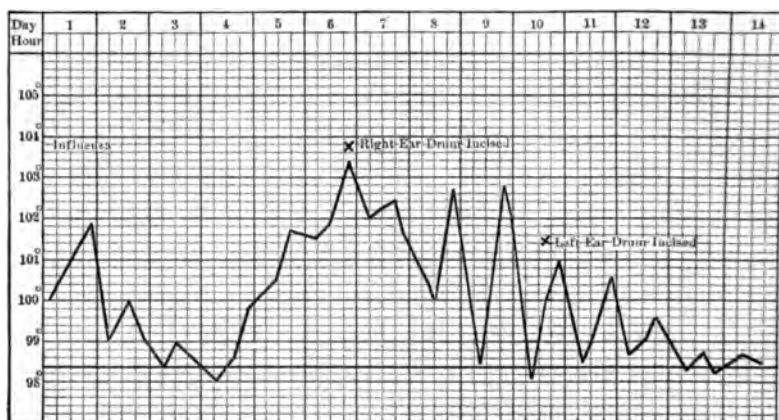


FIG. 178.—TEMPERATURE CHART OF ACUTE OTITIS FOLLOWING INFLUENZA, IN A CHILD THREE YEARS OLD.

days without outward evidences of pain or other signs of inflammation, and the discharge is the first symptom which leads the physician to suspect disease of the ear. In other cases there is marked dullness, apathy, anorexia, and sometimes nausea and vomiting, but for several days no evidence of pain; the temperature may be but little elevated. Thus, in most of the attacks seen in infancy, pain is not very marked, and it is this which so often leads to the great obscurity of the symptoms.

In older children the symptoms are more characteristic. Pain is usually sharp and severe, and is complained of early in the attack. The temperature is nearly always elevated two or three degrees, and occa-

sionally it is 103° or 104° F. (Fig. 179), with severe headache, extreme restlessness, and even delirium or convulsions, so that meningitis may be suspected.

The inflammation does not necessarily go on to suppuration and rupture. There are even more frequently seen, accompanying ordinary head-colds or mild attacks of influenza, cases in which the pain is quite severe for twenty-four or thirty-six hours, and accompanied even by a moderate elevation of temperature, and yet which rapidly subside without further symptoms.

In infants suffering from malnutrition or marasmus, otitis often comes on without any objective symptoms, the first thing noticed being the discharge.

Of all the symptoms, fever is the most constant, and is present in all cases except those just mentioned. The usual range of temperature is from 100° to 102° F.; exceptionally it may be from 103° to 105° F. The course

of the temperature is irregular. After spontaneous rupture or incision of the drum membrane the temperature usually falls, but often not immediately. Pain is more marked in older children than in infants—first, because in the latter the drum membrane is not so firm, yields more readily, and ruptures earlier; and, secondly, because the inflammation is usually of the catarrhal and not the phlegmonous type. Tenderness is sometimes elicited by pressure, especially just in front of the external auditory meatus; there may be increased sensitiveness of all parts of the ear and even of the whole side of the head; but no reliance should be placed upon the absence of such symptoms in excluding otitis. Children often complain of noises in the ear. Cerebral symptoms are infrequent, and occur chiefly in cases not receiving proper early treatment; they may indicate meningeal congestion, or, less frequently, localised meningitis or thrombosis.

In secondary otitis, especially when complicating severe scarlet fever, diphtheria, measles, or typhoid fever, all subjective symptoms are frequently wanting; unless the ears are examined the disease may be overlooked until rupture has taken place.

The local appearances in the early stage are marked redness and con-

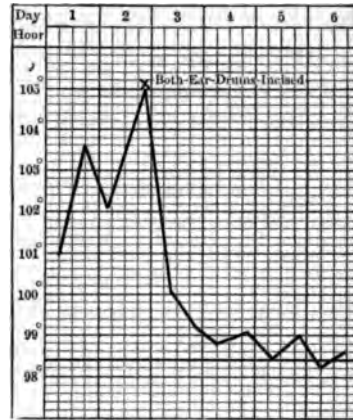


FIG. 179.—TEMPERATURE CHART OF ACUTE OTITIS ABORTED BY EARLY PARACENTESIS. Boy nine years old; attack followed a mild catarrh; severe pain in both ears began in afternoon of second day. Both drum membranes found acutely congested and bulging; incision followed by free hæmorrhage and immediate relief of pain. No suppuration occurred; patient well on fifth day.

gestion; later there is distinct bulging. If perforation has taken place, its site may or may not be visible, but its existence may be assumed if bubbles of air are seen deep in the canal, and if much mucus or pus is present, as inflammation of the external canal seldom causes a discharge. The pus sometimes burrows between the cartilages and escapes externally behind or at the side of the ear. In the catarrhal form the discharge is at first sero-mucus and quite profuse; later it is purulent. In the phlegmonous form it is always purulent, and liable to a sudden arrest with an increase in the constitutional symptoms.

**Diagnosis.**—Otitis in infancy is frequently obscure, because the patient is too young to direct attention to the seat of pain, or because the pain is slight or absent. The temperature is almost invariably elevated, and the usual problem presented is to discover a cause for this fever. The examination of the ears with a speculum should be done as a matter of routine in all children with fever, especially those in whom the cause of the fever is not perfectly clear. Otherwise many cases will be overlooked. A leucocytosis of 15,000 to 20,000 is almost invariably found. Local tenderness, deafness, or noises in the ears are significant when present, but are often wanting. Otitis is so common a cause of high temperature in infants during the cold season, that one should always have it in mind.

**Complications and Sequelæ.**—Remote consequences are most likely to be seen in cases following scarlet fever, probably because of their severity, particularly when early treatment has been neglected.

**Mastoiditis.**—This is the most frequent complication of acute otitis. In infancy the mastoid process is small and contains but a single cavity, the mastoid antrum, which communicates directly with the vault of the tympanum. It is probable that in every severe case of acute suppurative otitis there is some pus in the antrum. This is usually discharged into the middle ear after the tympanic membrane is incised or ruptures spontaneously. The principal cause of mastoid involvement is want of proper early treatment in acute otitis, particularly the practice of allowing these cases to take their natural course instead of securing early drainage by incision of the drum membrane.

The important symptoms of acute mastoiditis are fever, mastoid tenderness, and swelling. If mastoiditis develops rapidly after acute otitis the temperature may be high—103° to 105° F., and the leucocytosis is somewhat greater; if it develops gradually and appears late the temperature may be scarcely above 100° F. Abrupt cessation of an ear discharge should always arouse suspicion. It is always difficult to determine the presence of a slight amount of mastoid tenderness, but persistent tenderness of one side only is significant. It is often most marked close behind the auricle just over the antrum. The early swelling is due to œdema; later there may be an accumulation of pus. Post-

auricular abscess causes a very characteristic swelling, the ear standing out from the head. It is usually due to spontaneous rupture through the outer bony wall just over the antrum; it may occur where there has been no discharge from the ear. It is a frequent result of severe cases of acute mastoiditis not operated upon, especially in young children.

The characteristic otoscopic appearances of acute mastoiditis are: bulging of Shrapnell's membrane and drooping of the upper posterior wall of the external auditory canal.

*Meningitis.*—This is very rare in infants, but is more common in older children. There may be a localised pachymeningitis with the formation of pus—an epidural abscess—or, less frequently, general purulent meningitis. It may be secondary to other lesions, such as thrombosis of the lateral sinus, or the rupture of a cerebral abscess, but is usually due to infection through the roof of the tympanum, or along the internal auditory meatus. Meningitis may occur either with acute or chronic cases. Its symptoms are those of a severe acute meningitis; its duration is short; its termination almost invariably in death.

*Cerebral Abscess.*—This is due to a direct extension of the infection from the bone, veins, or dura mater. In about two-thirds of the cases the abscess is in the temporo-sphenoidal lobe. The next most frequent seat is the lateral lobe of the cerebellum. Körner states that disease of the mastoid and middle ear leads to cerebral abscess, and disease of the labyrinth to cerebellar abscess. Abscesses may be complicated by thrombosis or by meningitis. They are often latent until just before death, which more frequently occurs from the development of purulent meningitis than from any other cause. They are rare except in otitis of long standing.

*Thrombosis of the lateral sinus* may be simple or septic. In the former there is occlusion of the vessel by a fibrinous clot; in the latter there are in addition, micro-organisms.

Simple thrombosis causes no important symptoms. Septic thrombosis is relatively infrequent and causes very marked and severe symptoms. It follows operation upon the mastoid, or occurs as a complication of mastoiditis quite apart from operation. The temperature is usually of a high and widely fluctuating type, and there may also be chills. In some cases the constitutional symptoms, except fever, may not at first be severe, but may suddenly become very grave. Marked cerebral symptoms often develop rapidly, and death may follow in from twelve to twenty-four hours. At autopsy there may be found a soft broken-down clot in the sinus, which may extend into the jugular. It may be followed by secondary lesions of a general pyæmia, or by localised or general meningitis. Blood cultures usually give positive information.

*The labyrinth* is infrequently involved, although cases are recorded by Pye, Phillips, and others, in which the necrosis and discharge of the

entire labyrinth has occurred after scarlet fever. In most of these cases the deafness was complete, and in several vertigo was present.

*Facial paralysis* rarely occurs in the acute cases, but accompanies a considerable proportion of the chronic ones. It is especially seen in the tuberculous variety. It is due to an extension of the inflammatory process from the bone to the seventh nerve, where it passes through the canal. The symptoms are those of ordinary peripheral facial palsy.

**Treatment.**—Something may be done in the way of prophylaxis. It is of the first importance to secure a normal condition of the mucous membrane of the naso-pharynx by the removal of enlarged tonsils, adenoids, etc. The occasional attacks of earache accompanying these conditions are pretty sure to be followed by more serious trouble unless they are relieved. Whether during attacks of measles or scarlet fever, much can be done to prevent otitis, is still a mooted question. Personally I believe the risks of infection of the middle ear when judicious nasal syringing is employed are less than when nothing is done to cleanse the rhino-pharynx.

The medical treatment of acute otitis aims at the relief of pain and arrest of the inflammation. If the case is seen in the early stage the introduction of a few drops of a solution of adrenalin into the nostrils and ears and repeated every two or three hours, will sometimes abort an attack. This may be aided by free catharsis and the application of dry heat or frequent syringing of the ears with a saline solution as hot as can be borne. Neither oil nor laudanum should be dropped into the ear as is so often done in domestic practice; but there is no objection to a few drops of a four-per-cent solution of cocaine, or a five-per-cent solution of carbolic acid, either of which may relieve intense pain. If the child is not soon comfortable, an opiate should be given which may not only relieve pain, but may have a favourable influence upon the inflammation.

A continuance of pain in spite of these measures, with an increasing temperature, calls for operative interference. But a more reliable guide is the appearance of the drum membrane. If in addition to these symptoms there is mastoid tenderness immediate paracentesis of the drum membrane is imperative. An early incision is usually followed by a discharge of blood only; but tension is relieved, pain disappears, and the inflammation often quickly subsides without the formation of pus. (See Fig. 179.) Much suffering is thereby avoided; the wound rapidly heals, and much less damage is done than by allowing the disease to go on to a spontaneous rupture. Later incision may be required either for the relief of pain or for the evacuation of pus to prevent, if possible, the disease from spreading to the bony parts. The advantages of early paracentesis in acute otitis can hardly be overstated. Properly performed, it is free from risk, causes little or no shock, and should be advised in many

cases even in which the indications are not so clear as those above described. I favor incising the drum membrane in cases of doubt rather than waiting for more definite indications with the attendant risks of delay.

In the secondary otitis of scarlet fever, measles, and diphtheria, the indications for paracentesis are usually to be derived from the appearance of the drum membrane alone, other symptoms being absent or masked by the primary disease.

After incision or spontaneous rupture of the drum membrane the ear should be syringed every two or three hours with a warm solution of bichloride (1 to 10,000), or a saturated solution of boric acid, or simply with a sterile salt solution. A bulb ear-syringe of soft rubber or a fountain syringe may be used.

In most cases the discharge ceases in from one to three weeks; should it continue longer, some measures for checking it may be used. The use of a few drops of a 1 to 3,000 solution of bichloride in sixty-five per cent alcohol after syringing is of some value. It should be used with a medicine dropper. When the discharge has become foetid, syringing once a day with a solution of peroxide of hydrogen (1 to 2) is often useful. A persistent discharge often depends upon the fact that the child's general condition is poor, and improvement in this is more important than any variation in local treatment.

When symptoms pointing to acute mastoiditis are present, early free incision of the drum membrane is indicated, and a mastoid ice-bag should be applied continuously for thirty-six or forty-eight hours. In addition, in older children, the artificial leech may be placed over the antrum or the mastoid tip. With these measures the inflammation often subsides. Regarding operation upon the mastoid, my own belief is that it is now performed too frequently and with insufficient indications, especially in infancy and early childhood. The operation is a serious one, and at this age its immediate risks are considerable. I have personally known of a number of deaths directly connected with it, and of others occurring at a later period, where the child was worn out by the long after-treatment, dying perhaps from some intercurrent disease or from exhaustion. On the other hand, the dangers to which patients are exposed who are not operated upon have, I think, been greatly exaggerated. In my own experience, meningitis, sinus thrombosis, and cerebral abscess do not occur in anything like the proportion of cases that the surgeons would have us believe.<sup>1</sup>

While I fully appreciate the value of the operation, and am quite sure that lives are often saved by its timely performance, I would in-

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<sup>1</sup> The records of the New York Foundling Hospital, with a resident population of about 800 infants and young children, showed 573 cases of acute otitis in five years (1900 to 1904 inclusive). During this period there were three extensive epidemics of measles with a total of 1,034 cases; 166 cases of scarlet fever; 578 cases of diphtheria;

sist that it be done only with very positive and clear indications. In infants, localised tenderness is difficult to determine; and fever after acute otitis may be due to many other conditions. In very young patients we should therefore insist upon other symptoms before deciding to operate. The risks of waiting for clearer indications are, I believe, much less than those attendant on unnecessary operation. Often the cause of the temperature is found in the lungs; and not very infrequently a moderate pulmonary congestion or bronchitis becomes a pneumonia as a consequence of the prolonged anæsthesia necessary for the operation. With infants therefore in case of any doubt, as to diagnosis or the progress of the case, one should invariably decide against operation, or at least for postponement. With older children, however, conditions are somewhat different; diagnosis is easier and the operative risk much less.

The treatment of chronic otitis and of the associated conditions is largely surgical, and belongs to the specialist; but it is extremely important that the general practitioner should be familiar with their symptoms, and realise the danger from these neglected cases, not only to the function of hearing, but also to life itself. The essential thing in treatment is that the operation should be thorough enough to secure free drainage, and to permit thorough cleansing of the parts. Too much can not be said against the expectant treatment of these cases, or against the practice of prolonged poulticing.

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and 1,505 cases of pneumonia. With the 573 cases of otitis, acute mastoiditis was recognised and recorded in but 17 patients. It is not improbable that other mastoid inflammations were overlooked. In this institution, however, nearly every fatal case comes to autopsy, and if an unrecognised mastoiditis had led to a fatal result the autopsy records should show it. In the five-year period, 900 autopsies were made. There was no instance recorded of abscess of the brain following otitis. There were but two examples of acute meningitis following otitis with mastoiditis; but there were 14 cases of acute meningitis secondary to other conditions—pneumonia, 10; to pericarditis, 2; to empyema, 1; to diphtheria, 1. During the period mentioned there were 11 mastoid operations performed in the hospital, with 6 recoveries and 5 deaths, all from causes directly connected with the operation.

If mastoiditis follows otitis, complicating the acute infectious diseases of early childhood as often as has been claimed, we must admit that a very large proportion of the patients may get well without operation.

## SECTION IX.

### THE SPECIFIC INFECTIOUS DISEASES.

ACCURATE classification of the infectious diseases is at the present time impossible, but there are two quite distinct groups into which, with one or two exceptions, those here considered may be placed.

The first group includes scarlet fever, measles, rubella, and varicella. The nature of the specific poison in each of these is as yet unknown. They are, strictly speaking, contagious; for it is practically certain that any of them may be contracted by proximity to a person suffering from the disease, without actual contact. In no one of these diseases is the poison given off in a single definite discharge, and in no one is there a characteristic visceral lesion. These peculiarities, together with the fact that thus far the poison of each of these diseases has resisted all attempts at isolation, render it not improbable that the exciting cause in each is some other variety of micro-organism rather than a bacterium.

In the second group may be placed diphtheria, pertussis, influenza, typhoid fever, and tuberculosis, in each of which the specific poison is a known form of bacterium. Each of these diseases, except pertussis and influenza, is associated with definite and characteristic visceral lesions. The poison is discharged from the body in a certain well-understood manner from the tissues which are affected by the disease, and in no other way.

Syphilis and malaria have not been included in either of the above groups. They belong in a class by themselves.

## CHAPTER I.

### SCARLET FEVER.

(*Scarlatina.*)

SCARLET FEVER is an acute, contagious, self-limited disease, one attack usually protecting the individual through life. The period of incubation is usually from two to four days; that of invasion, from twelve to twenty-four hours; that of eruption, from four to six days; that of desquamation, from three to six weeks. The disease may be communicated at any time from the first symptom of invasion throughout des-



quamation, and even during the existence of purulent discharges from the nose or other mucous or serous membranes. It is usually ushered in by vomiting, fever, and sore throat, and is characterised by an erythematous rash appearing first upon the neck and spreading rapidly over the entire body. Its chief complications are otitis, adenitis, and membranous inflammations of the pharynx, which frequently extend to the nose, rarely to the larynx. The most important sequelæ are otitis and nephritis. The constancy of the throat infection in scarlet fever strongly points to the pharynx as the point of entry of the infection.

**Etiology.**—Analogy leads to the belief that scarlet fever is due to a micro-organism, but as yet its nature has not been discovered. The complications are usually associated with the development of a streptococcus. Some have gone so far as to claim that a streptococcus is the cause of the disease. From present knowledge, however, it appears rather to play the rôle of a secondary or accompanying infection, for the development of which the mucous membranes of a person suffering from scarlet fever seem to afford most favourable conditions. To the streptococcus may be ascribed the membranous inflammations of the tonsils and pharynx, the otitis, the inflammation of the lymph nodes and the cellular tissue of the neck, and probably also the nephritis, endocarditis, pneumonia, and joint lesions. In many of the above conditions the streptococcus is associated with other pyogenic germs, and in some cases with the diphtheria bacillus. The exact rôle played by the streptococci and by the virus of scarlet fever in these complications is still a matter of dispute, the probabilities being that some are due to one and some to both of these infective agents.

**Predisposition.**—The susceptibility of children to the scarlatinal poison is much less than to that of measles; still, it is much greater than that of adults. Billington (New York) records observations made in twenty-six families living in tenements where little or no attempt at isolation was made. In these families there occurred forty-three cases of scarlet fever; but forty-seven other children, although unprotected by previous attacks and constantly exposed, did not contract the disease.

Johannessen reports that of 185 children under fifteen years who were exposed, twenty-eight per cent contracted the disease; while of 314 adults, only five per cent contracted the disease. It may be stated that, approximately, not more than one-half of the children exposed take the disease. The susceptibility is not great in early infancy, but it increases until about the fifth year, after which it steadily diminishes. Both sexes are equally liable to scarlet fever. Epidemics are more frequent in the fall and winter than in summer, and cases occurring in the cold months are apt to be more severe. Whitelegge, in 6,000 cases, found the highest mortality in the month of October; and in Caiger's report of 1,008 cases this was also the month showing the greatest mortality.

*Incubation.*—Of 113 cases<sup>1</sup> in which the period of incubation could be accurately determined, it was as follows:

|                            |          |                  |            |
|----------------------------|----------|------------------|------------|
| 24 hours or less . . . . . | 6 cases. | 8 days . . . . . | 2 cases.   |
| 2 days . . . . .           | 15 "     | 9 " . . . . .    | 5 "        |
| 3 " . . . . .              | 28 "     | 11 " . . . . .   | 1 case.    |
| 4 " . . . . .              | 25 "     | 14 " . . . . .   | 1 "        |
| 5 " . . . . .              | 6 "      | 21 " . . . . .   | 1 "        |
| 6 " . . . . .              | 15 "     |                  |            |
| 7 " . . . . .              | 8 "      |                  |            |
|                            |          |                  | 113 cases. |

Thus in eighty-seven per cent of these it was between two and six days, and in sixty-six per cent between two and four days. Speaking generally if, after exposure, a week passes without symptoms, the chances of infection are very small. A short incubation is more frequently seen in severe than in mild cases.

*Mode of Infection.*—The chief source of infection is the patient himself. It is somewhat doubtful whether the poison of scarlet fever can be conveyed by the breath, but it surely is by discharges from the mucous membranes involved, probably by the scales during desquamation, and by all the excretions of the patient—urine, fæces, and perspiration. Infection often takes place from the carpets or furniture of the sick-room, and from the clothing of the patient. In a city, the bedclothing, while airing in the window, has been known to convey the disease to an adjoining house. Instances are recorded of the spread of scarlet fever by the washing of infected with other clothing. Toys or books may be carriers of the disease. A bouquet of flowers sent from a sick-room to an institution, in one instance proved a vehicle of infection. Cats, dogs, and other domestic animals are known to have conveyed the disease. Scarlet fever is sometimes spread by food, particularly by milk.

The transmission of the disease through a third person is not frequent, but numerous instances of it are on record. The persons most likely to carry it are the nurse and the physician. Physicians have in many cases carried scarlatina to their own children, but only when there had been very direct contact with the patient, and where the interval before seeing the second child was short. The clothing of the nurse may be almost as infectious as that of the patient. The transmission of the disease by one who, although living in the house, does not come in contact with the patient is extremely improbable. An instance is recorded in Allbutt where scarlatina was transmitted through two healthy persons.

*Duration of the Infective Period.*—There is no evidence to show that the disease is communicable during the period of incubation. It is

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<sup>1</sup> Part of these are from personal observation, but the great majority are isolated cases scattered through medical literature, occurring under circumstances which made it possible to determine the exact length of the incubation period.

slightly contagious from the beginning of invasion, before the rash appears. Infection appears to be most active at the height of the febrile period—from the third to the fifth day—and, next to this, during the stage of active desquamation.

In simple cases, the average duration of the contagious period may be placed at six weeks, or until desquamation is complete. However, physicians generally have been accustomed to place too much stress upon the danger from the scales, and too little upon that from the discharges from the mucous membranes. Early infection comes chiefly from the throat, nose, or possibly the breath. Late infection may arise from a purulent otitis, rhinitis, chronic pharyngitis, suppurating glands, empyema, and possibly also from the urine in nephritis. The infectious nature of these purulent discharges has not been sufficiently recognised. It is possible for them to convey the disease during a period of several months. One case is recorded in which scarlatina was communicated through a purulent nasal discharge after eleven weeks; another in which the opening of a post-scarlatinal empyema in a surgical ward was followed by an outbreak of scarlet fever.

In winter especially, a chronic pharyngeal catarrh may long contain the germs of infection. Ashby found, on careful investigation, that from two to four per cent of patients discharged from a scarlet-fever hospital subsequently conveyed the disease. There is particular danger from a child who has recently had the disease sleeping with other children. Line records a case in which this was the means of conveying the disease after fourteen weeks, and when the patient had been considered perfectly well for three weeks. It is impossible to say that at any specified time absolute safety exists. All patients before being discharged from a hospital or released from quarantine in private practice, should be carefully examined as to the condition of the mucous membranes, and quarantine continued as long as catarrhal inflammations are present. The poison of scarlatina clings more tenaciously to clothing, upholstery, and apartments than that of any other infectious disease, possibly excepting tuberculosis. Authentic cases are on record in which more than a year had elapsed between the first and second cases, where the source of infection seemed certain.

**Lesions.**—The only characteristic lesions of scarlatina are those of the skin and the mucous membranes of the mouth and throat. The skin is the seat of an acute dermatitis of variable depth and intensity. There is first acute hyperæmia, followed by an exudation of serum and cells in the corium, especially about the blood-vessels and hair follicles. There results a death of the epidermis which is thrown off in the desquamation. The mucous membrane of the mouth, tongue, and throat is the seat of a catarrhal, membranous, or gangrenous inflammation which rarely invades the larynx, but very frequently the middle ear and nose. The entire

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oesophagus is often the seat of an intense congestion. From the ear the infection may extend to the mastoid cells, the meninges, or the brain, and from the nose to the accessory sinuses, particularly the antrum of Highmore. All the lymph nodes about the neck may be involved, the infection ending in cell-hyperplasia, suppuration, or necrosis. The cellular tissue of this neighbourhood may also become infiltrated, this being followed sometimes by suppuration and occasionally by gangrene.

The most constant change throughout the body, according to Pearce, is hyperplasia of the lymphoid tissue, which is seen everywhere. The other lesions are degenerations due to the scarlatinal poison alone, or in conjunction with the various forms of secondary infection, or to the latter alone. The most important are: fatty degeneration of the heart; areas of focal necrosis in the liver; acute degeneration of the kidney or acute diffuse nephritis; proliferation of the cells of the Malpighian bodies of the spleen; broncho-pneumonia, gangrene, or abscess of the lung; pleurisy, which is often purulent; endocarditis, pericarditis; abscesses in the cellular tissue and inflammation of the joints. These visceral changes will be considered more fully under Complications.

**Symptoms.**—*Invasion.*—As a rule, the invasion of scarlet fever is abrupt, the symptoms at the onset usually being directly in proportion to the severity of the attack. In the majority of cases there is vomiting, a rapid rise in temperature, and soreness of the throat. Often the vomiting is repeated; it is frequently forcible, and without nausea. In severe cases the rise in temperature is very rapid, to 104° or 105° F.; in the mildest cases it may not be above 101° F. A child may complain of soreness of the throat, or the throat symptoms may be entirely objective. In most severe cases, there is a uniform erythematous blush covering the pharynx, tonsils, and fauces, but on the hard palate there are minute red points. The appearance of this is usually coincident with the rise in temperature. Occasionally membranous patches may be seen upon the tonsils the first day, but not generally before the third or fourth day. In mild cases the throat shows only a very moderate congestion. Severe cases are sometimes ushered in by convulsions, especially in very young children. Diarrhoea is not uncommon in summer. There is general prostration, which is directly proportionate to the height of the fever.

*Eruption.*—This usually appears from twelve to thirty-six hours after the first symptoms of invasion; exceptionally, not until the third or even the fifth day. A later appearance than this is somewhat doubtful, for the rash not infrequently recedes and reappears, having been overlooked in the first instance. In 108 cases observed in the New York Infant Asylum, the duration of the rash was as follows:

|                            |          |
|----------------------------|----------|
| Two days or less .....     | 5 cases. |
| Three to seven days .....  | 81 "     |
| Eight to eleven days ..... | 16 "     |
| Over eleven days .....     | 4 "      |
| Recurring .....            | 2 "      |

These statistics are confirmed by the observations of most writers, that the rash lasts from three to seven days. The full development of the rash is generally seen in from twelve to twenty-four hours from its first appearance, and not infrequently the whole body is covered in the course of four or five hours. Very rarely its extension is so slow that it is two or three days before the body is covered. Its first appearance is almost invariably upon the neck and chest. In the cases of moderate severity the typical rash is seen. Its colour is red rather than scarlet, and on close inspection it is seen to be made up of very minute points upon a reddish ground giving the appearance of a uniform blush; or the background may be wanting and only the punctate eruption shows. These points are the papillæ of the skin and hair follicles. The rash usually covers the entire body except the face. This may be the seat of an ordinary flush. Even in cases with intense eruption the central part of the face usually escapes, though elsewhere the eruption may be as bright as upon the body. There is often a peculiar pallor about the mouth which is characteristic. The appearance of the eruption in dark-skinned races is much modified and often difficult of recognition. In the negro the palms and soles may be the only places where the eruption can be distinguished. Here may be seen a bright red blush or a fine papular eruption.

Variations in the eruption are very frequent and very puzzling. They occur especially in the very mild and in the most severe cases.

In the mild cases the rash is not seen upon the face; it is often faint upon the body, and may be present only upon certain parts; when the rash is faint or scanty it is usually most marked in the groins and axillæ, or over the buttocks and back of the thighs; it may last only one day, and sometimes may be so slight as to escape notice altogether. It may be absent in some very mild cases, in certain others where the throat symptoms are severe, and in malignant cases. In the very severe cases many irregularities are seen, both as to the time of the appearance of the eruption and its character. Sometimes it occurs as large, irregular patches; again, it is macular, closely resembling the rash of measles; occasionally it is of a dark purplish colour; and very rarely it is hæmorrhagic. Not infrequently an eruption of fine vesicles is seen, especially on the chest, axillæ, and abdomen. It is seen both in mild and severe cases. This is especially diagnostic. A well-developed bright rash indicates strong heart action, and a sudden recession of the rash is a sign of heart failure. Often a rash which is faint and doubtful in character may be brought out fully by a hot bath.

With the eruption at its height, there is intense itching or burning of the skin, and in severe cases considerable swelling, chiefly noticeable upon the hands and face.

*Desquamation.*—Shortly after the rash has faded, about the eighth day, there begins an exfoliation of the dead epidermis, known as desquamation. This is even more characteristic of the disease than is the rash. It is usually first seen upon the neck and chest, where it appears as fine flakes. The desquamation of the trunk is completed in from one to three weeks. If baths and inunctions are being used, it is scarcely perceptible. It continues longest where the epidermis is thickest—viz., upon the hands and feet—and here it lasts from four to seven weeks, and not infrequently eight weeks. The appearance of the fingers and toes during desquamation is characteristic. The finger tips usually peel first, and the new epidermis is pink and fresh-looking, while that which has not yet separated is of a dull gray colour and loosened at the margin. Occasionally the epidermis of a considerable part of a finger may be loosened at once, so that a partial cast may be thrown off like the finger of a glove. Sometimes the patient comes under observation for the first time during desquamation, the history of the early symptoms being doubtful or absent. Such desquamation as has been described, occurring both upon the hands and feet, may be regarded as conclusive evidence of scarlet fever, no matter what the history may be. In rare instances desquamation may include loss of the hair and the nails.

1. *The Mild Cases.*—The symptoms may be so slight as to be entirely overlooked, nothing being noticed until desquamation occurs. Usually, however, there is a rather abrupt invasion, with vomiting and a temperature from 100° to 103° F. The tonsils and pharynx are congested, while the palate shows a punctate redness somewhat like the cutaneous eruption. The papillæ of the tip and borders of the tongue are enlarged. Nearly always within twenty-four hours the rash makes its appearance, generally first upon the neck and chest. Very often it is not seen upon the face, but is abundant on the rest of the body. The rash fades on the third or fourth day, and has disappeared by the fifth day. There is very little prostration, the child often being with difficulty kept in bed.

The highest temperature is coincident with the full eruption, and is usually seen during the first thirty-six hours of the disease. It gradually falls to normal by the third or fourth day. Some examples are shown in Fig. 180. In the mildest cases the temperature may never be above 100° F.

Desquamation is often faint over the body, but is unmistakable over the hands and feet. It begins about the end of the first week, always being most marked where the eruption has been most intense.

The mild cases are usually uncomplicated, but the possibility of otitis and of late nephritis should always be kept in mind, as these may occur

even with the mildest attacks. The difficulties in diagnosis in mild attacks of scarlet fever are often great. It should be remembered that these cases are just as contagious as severe ones, and that from a mild attack a severe one is often contracted. It is frequently by these mild cases that this disease is spread in schools. In dispensaries I have often seen patients desquamating from scarlet fever, who had been attending

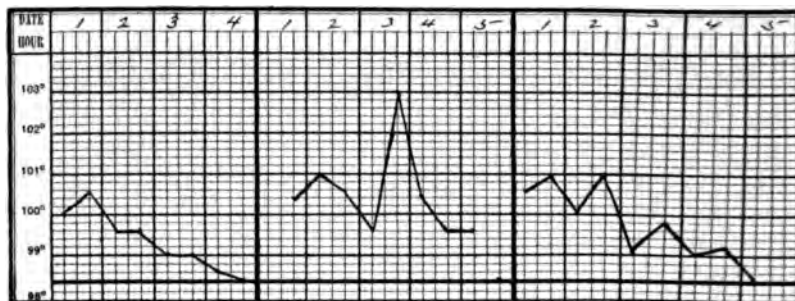


FIG. 180.—MILD SCARLET FEVER. Three cases occurring successively in the same family. Diagnosis not made until the third case developed, at which time the first one was found to be desquamating in a typical manner.

school regularly up to the time when they were brought for treatment for nephritis or some other disease.

2. *Cases of Moderate Severity.*—The onset is sudden with vomiting, which is usually repeated, rarely with convulsions. The temperature rises rapidly, and by the end of the first twenty-four hours has reached 104° or 105° F. The rash generally appears within the first twenty-four hours, and its intensity is usually in direct proportion to the severity of the attack. Appearing first upon the neck or chest, it extends rapidly, covering the entire trunk and extremities, often in a few hours. It is generally typical in appearance, being made up of minute points, but giving the appearance of a uniform blush, which has been compared to a boiled lobster. Little change takes place in the rash for four or five days. After this it fades quite rapidly, and disappears by the sixth or seventh day.

The throat resembles that of the mild form, except that the redness is more intense and there is slight swelling of the tonsils, fauces, and uvula, and often pain upon swallowing. Occasionally small yellowish patches are seen upon the tonsils by the second or third day, but these can be wiped off and are not distinctly membranous. There is usually a moderate discharge of a sero-purulent character from the nose. The lymph glands at the angle of the jaw are swollen and quite tender. The tongue may be coated in the centre and show bright red points at its borders and tip, or it may be quite red and show the prominent papillæ everywhere—the “strawberry tongue”; while not exclusively seen in

scarlatina, this is of considerable diagnostic value. It is rarely seen before the third day, and may continue several days or even weeks.

During the height of the fever there is restlessness, thirst, and not infrequently slight delirium. The temperature usually reaches the maximum by the second day, and falls gradually, but even in uncomplicated cases the fever often lasts from ten to fourteen days (Fig. 181). The pulse in the early part of the disease is rapid, its frequency being usually out of proportion to the height of the temperature. There is much prostration, frequently followed by quite a marked degree of anæmia.

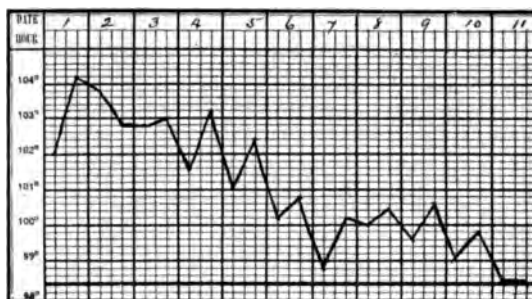


FIG. 181.—TYPICAL TEMPERATURE CURVE OF UNCOMPLICATED SCARLET FEVER OF MODERATE SEVERITY. Girl three years old.

This form of the disease rarely proves fatal apart from complications. The complications seen most frequently in this form of scarlet fever are adenitis, otitis, and pneumonia. Nephritis is the only common sequel.

3. *The Severe Cases.*—The severe type of scarlet fever usually declares itself from the beginning. The incubation is short, and the full rash may be seen within a few hours after the initial symptoms. It is usually intense and covers the entire body, even including the face. In other cases the eruption is delayed, often scanty, and may disappear in a few hours. The disease assumes one of two fairly distinct types; one is characterised by the severity of the general toxæmia, the other by the predominance of the throat symptoms. In the first group the toxæmia is shown by the height of the temperature, the severity of the nervous symptoms, and the profound cardiac depression. The temperature quickly rises often to 105° or 106° F., and usually remains steadily high until the death of the patient. The nervous symptoms are great prostration and delirium, which is sometimes active, but more often low and muttering. The pulse is very rapid, 160 to 180 being not uncommon; it is weak, compressible, often irregular, and the muscular sounds of the heart are feeble. The urine is scanty and almost invariably albuminous. Hæmorrhages from the mouth, the nose, or other mucous membranes are occasionally seen. The duration of the disease in this form is generally from five to seven days. Exceptionally the symptoms develop with greater intensity, and death follows in three or four days. A shorter duration than this, the so-called malignant scarlet fever, is exceedingly rare.

In the second group with predominant throat symptoms the first



three or four days may show nothing more than cases of the moderate type. Membranous patches appear upon the tonsils and spread to the soft palate, uvula, and pharynx, sometimes to the nose and through the Eustachian tube to the ear, very rarely involving the larynx. The mucous membrane of the mouth is intensely congested, and often partly covered by membrane; there are sordes on the lips and teeth, and there may be superficial ulcers, which bleed readily. The glands of the neck swell rapidly, often to a great size, and the cellular tissue about them is infiltrated. The head is thrown back to relieve the dyspnoea which the pressure from this swelling occasions. There is an abundant discharge from the nose and mouth; the breath is very offensive. The general symptoms are those of a severe septicæmia. The temperature is steadily high, usually between 103° and 105° F., for about a week, after which in cases ending in recovery it slowly falls unless complications develop (Figs.

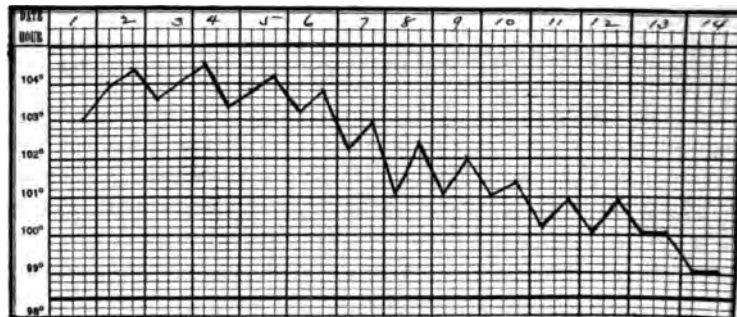


FIG. 182.—TYPICAL TEMPERATURE CURVE OF SEVERE SCARLET FEVER ENDING IN RECOVERY. Prolonged course due to severe throat symptoms lasting from second to sixth day, otherwise uncomplicated; boy twelve years old.

182, 184, 185). But even in uncomplicated cases the fever sometimes continues for three weeks. In fatal cases the temperature may be steadily high till death (Fig. 183), or it may fluctuate widely. The pulse is rapid, weak, and irregular. There is complete anorexia; both food and stimulants may have to be given by gavage. There is low delirium or apathy, and sometimes all the symptoms of the typhoid condition are present.

Signs of a broncho-pneumonia may be found in the chest, and by the end of the first week or early in the second, acute otitis often develops. The urine is rarely free from albumin, but the amount present is not usually great; there may be hyaline and epithelial casts, and sometimes blood. In some cases the throat symptoms predominate; in others, those of general sepsis, but more frequently the two are combined and are directly proportionate to each other. In still other cases, instead of the membranous inflammation, it may be of a gangrenous character, and extensive sloughing may take place in the pharynx or the cellu-

lar tissue of the neck, sometimes exposing or even opening the great vessels.

The duration of the symptoms in cases with severe angina is from seven to fourteen days. There is increasing prostration and finally a septic stupor, with death from exhaustion, from heart failure, or from

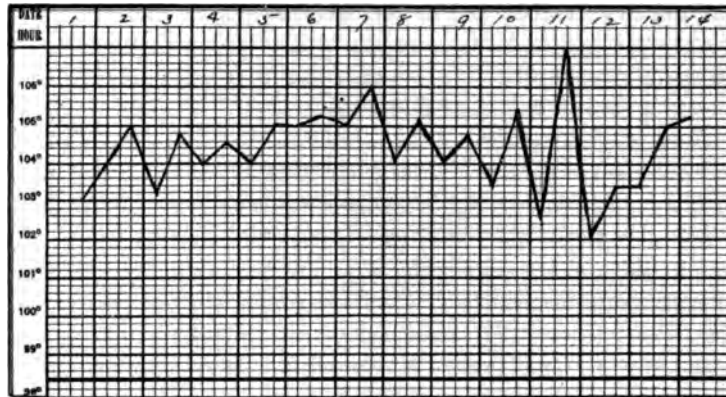


FIG. 183.—SEVERE SCARLET FEVER, SEPTIC TYPE; DEATH ON FOURTEENTH DAY. Intense angina; otitis; nephritis; necrotic inflammation of cervical lymph glands; girl seven years old; death from heart failure.

some complication—broncho-pneumonia, pleurisy, nephritis, hæmorrhages following sloughing, pericarditis, or endocarditis. In cases which recover, the acute symptoms nearly always continue for a full month; and after escaping the dangers of sepsis and the early complications, the child has still to run the gauntlet of all the late complications—nephritis, pneumonia, endocarditis, pyæmia, etc. A case may prove fatal as late as the end of the seventh week; nearly all such results are due to nephritis or to its complications.

4. *Surgical Scarlet Fever.*—The existence of a special form of scarlet fever occurring in patients with recent wounds or those who have been subjected to surgical operations, while stoutly maintained by several writers, has been vigorously denied by others. The question is one difficult of solution on account of the close similarity at times existing between the symptoms of scarlet fever and sepsis, and the necessity of deciding in an undoubted case whether the infection with scarlet fever was dependent upon or coincident with the wound.

Hamilton, from a study of 174 reported cases, reached the conclusion that proof of the existence of a special form of scarlet fever rests upon the reports of cases usually meagre, and careful analysis of these would lead one to consider them rather as septic than as scarlatinal infections; that when there was undoubted evidence of scarlet fever, there was no proof that it was in any way due to the coincident wound, and that there

is as yet no convincing proof in the literature that surgical scarlet fever is anything more than scarlet fever in the wounded. On the other hand, there have been observed clinically cases which seem to admit of no other reasonable explanation than that an abrasion of the skin, a recent wound, or even possibly a varicella vesicle, may be the point of entry of the scarlatinal infection, instead of the more usual portal, the pharynx.

**Relapses, Recurrences, and Second Attacks.**—As a rule, one attack of scarlatina gives immunity through life. The exceptions are very few, but are well authenticated. I have seen but once an undoubted instance of a second attack in the same individual.

Relapses or recurrences within a brief period after the first attack are more frequent. There are to be excluded the cases of pseudo-relapses in which the rash, having temporarily subsided for two or three days, reappears; also those where the rash varies in intensity from time to time; and, lastly, the cases in which, occurring late in the disease, it is due to septicæmia or pyæmia. They are comparable to the relapses of typhoid fever. They occur most frequently during desquamation, between the seventh and twenty-fourth days. There may be not only a new eruption, but a rise of temperature, sore throat, and vomiting, just as in the initial attack. These recurrences are sometimes shorter and milder than the first attack, but this is by no means uniform, since Körner mentions eight cases where the second attack proved fatal.

In considering the subject of second attacks, the liability to errors in diagnosis must be borne in mind and only cases included which have presented typical symptoms.

**Special Symptoms, Complications, and Sequelæ.**—*Temperature.*—The temperature curve of this disease is quite characteristic. There is usually seen an abrupt rise, the maximum being reached on the second day; there follows a period of variable duration, generally lasting, according to the severity of the case, from two to five days, in which the fluctuations are very narrow; then a gradual decline to normal, which is reached in the milder cases in about a week; in those which are more severe, in about two weeks. This typical curve (Figs. 179 and 180) is seen in the great proportion of uncomplicated cases which end in recovery. Deviations from it, therefore, are important as indicating that some complication exists. The explanation is usually to be found in the development of otitis, adenitis, nephritis, pneumonia, etc. Severe throat symptoms prolong the temperature but do not usually modify its course. In very severe cases ending fatally the high temperature is prolonged. In any case, a rise after the third day is unfavourable.

*Throat.*—Three distinct forms of angina are seen in scarlatina: simple or erythematous, membranous, and gangrenous.

1. Erythematous Angina.—This can hardly be ranked as a complication, as it is nearly as constant as the scarlatinal rash. Usually

there is only the intense general blush over the entire pharynx with the fine red points upon the hard palate; but there may be seen upon the tonsils grayish-yellow spots resembling those of follicular tonsillitis, which can be wiped off, leaving a clean surface. This simple angina is at its height with the maximum temperature, and fades as the temperature falls. It does not often extend to adjacent mucous membranes.

2. Membranous Angina.—These cases were formerly classed as scarlatinal diphtheria, and whether this process was identical with primary diphtheria or not, was for a long time a subject of much discussion. Cultures have shown that the great majority of these inflammations are not true diphtheria, but are due to the streptococcus.

The lesions of this form of angina are considered in the chapter on Membranous Tonsillitis. Usually on the second or third day of the disease an exudation appears upon the tonsils, and in the milder cases it covers only the tonsils. In the most severe form it may be seen within twenty-four hours of the onset, sometimes before the eruption appears. Beginning upon the tonsils, the membrane rapidly spreads to the entire pharynx, the mucous membrane of the nose, the mouth, the Eustachian tube, and even to the middle ear. In colour it may be gray, greenish, or almost black. There is so much swelling of the throat that swallowing becomes difficult. The infiltration of the cellular tissue of the neck and the enlarged lymph glands produce great external swelling, which may extend like a collar from ear to ear. The breath has a foul odour, the nasal discharge is thin and foetid, and nasal respiration is obstructed, so that the mouth is open constantly. It is surprising that the larynx is so seldom invaded.

These local changes are accompanied by constitutional symptoms of great severity, which are due to a general streptococcus septicæmia; broncho-pneumonia and nephritis are very frequent, otitis is almost constant, and suppuration of the lymphatic glands is not uncommon. The eruption is often irregular and late in appearing.

The frequency with which diphtheria coexists with scarlatina varies greatly. In hospital practice the proportion often runs as high as thirty or forty per cent. In private practice it is much lower. In some epidemics it is much more frequent than in others. The streptococcus angina is usually seen at the height of the disease; true diphtheria may occur at any time, even during convalescence. Very little reliance is to be placed upon the appearance of the membrane. The only positive means of differentiation is by cultures, which should invariably be made from the throat of every patient admitted to a scarlet-fever hospital, and of every case in private practice showing any exudate upon the tonsils. If the first culture is negative and the throat symptoms increase, repeated cultures should be made.

3. *Gangrenous Angina*.—This is seen only in the worst cases of scarlet fever. The process may be gangrenous from the outset, or precede by a membranous inflammation. It is sometimes insidious in its development. There is a foetid odour to the breath, an irritating discharge from the nose and mouth, with very great glandular swelling. The tonsils are gray or grayish-black in colour, and large masses of necrotic tissue may be removed with the forceps from the tonsils, uvula, fauces or pharynx, and sometimes sloughing occurs in the cellular tissue of the neck. Blood-vessels of considerable size are sometimes opened, and serious or even fatal hæmorrhage may result. The constitutional symptoms are those of great asthenia, prostration, and profound cachexia followed almost invariably by a fatal termination.

*Lymph Nodes*.—These are swollen in all cases accompanied by severe angina. The inflammation may be simply an acute hyperplasia, or may go on to suppuration and necrosis. Abscess does not often occur at the height of the disease, but the early swelling may almost completely subside only to recur, and suppuration may take place even as late as the fifth or sixth week of the disease. It may be confined to the glands or be complicated by suppuration in the cellular tissue of the neck.

*Cellulitis of the Neck*.—This usually occurs toward the end of the first week, and is associated with grave throat symptoms. Rapid and extensive infiltration occurs, the skin becomes tense and brawny, the head is held back, and there may be considerable dyspnoea. The infiltration may be only in the neighbourhood of the lymph glands or may be diffuse. Unless relieved by early incision, the diffuse form may result in suppuration and extensive sloughing, which may be deep enough to lay bare the large vessels of the neck. This is a complication of the gravest possible import. Death may occur from septicæmia before or after sloughing or from hæmorrhage due to opening by ulceration of the external carotid or some of its branches; or there may be associated thrombosis of the jugular vein, leading to thrombosis of the lateral sinus, meningitis, or pyæmia.

*Ears*.—The otitis is due to direct extension of the infection from the rhino-pharynx. It is the most frequent complication of scarlatina and in doubtful cases may have some diagnostic importance. As a rule the younger the child the greater the liability to otitis. It is more frequent in winter than at other seasons, and is closely connected with the severity of the throat symptoms. In an epidemic occurring in the New York Infant Asylum in spring and summer there were 73 cases of scarlatina and not one of otitis. In a fall and winter epidemic in the same institution two years later, of 43 cases 20 per cent had otitis. Of 4,397 cases reported by Finlayson, otitis occurred in 10 per cent, and of 1,008 cases reported by Caiger, in 13 per cent. In Burkhardt's statistics the

proportion was as high as 33 per cent. Of cases accompanied by severe throat symptoms otitis is present in fully 75 per cent.

As a rule, both ears are affected. Otitis is most frequent early in the second week, but may occur at any time, even during convalescence. In the cases when it develops at the height of the disease there are in some cases no new symptoms; in others there is pain and deafness and a rise in the temperature, which may fall after paracentesis or rupture of the drum membrane, or there may be extension to the mastoid (Fig. 184).

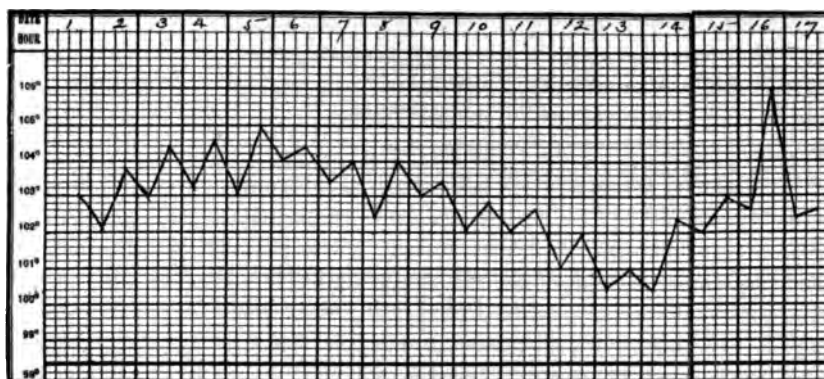


FIG. 184.—SEVERE SCARLET FEVER; OTITIS; MASTOIDITIS; DEATH. Typical symptoms and temperature curve until fourteenth day; secondary rise of temperature from otitis; double paracentesis on the fifteenth day; mastoid operation on the sixteenth day; death twelve hours later from septicæmia; boy five years old.

The otitis is often overlooked unless the ears are regularly examined. The form of inflammation may be catarrhal or purulent, the latter being often accompanied by necrotic changes.

Bezold makes the following report upon 185 cases showing the disastrous consequences of scarlatinal otitis: "In 30 there was entire destruction of the membrana tympani; in 59 the perforation comprised two-thirds or more of the membrane; in 15 there was total loss of hearing on one side, and in 6 of the cases upon both sides; in 77 of the cases the hearing distance for low voice was less than twenty inches."

As a cause of permanent deafness and deaf-mutism, no disease of childhood compares in importance with scarlet fever. May has collected statistics of 5,613 deaf-mutes, of whom 532 owed their condition to otitis following scarlet fever.

**Kidneys.**—Albuminuria accompanies nearly all the severe cases of scarlet fever. In many this is simply the ordinary febrile albuminuria due to acute degeneration of the kidneys. In those with severe throat complications, and in nearly all the septic cases, there is an acute diffuse nephritis; the interstitial changes may be very marked and the kidneys contain minute abscesses. This occurs at the height of the febrile process

and is rarely accompanied by dropsy; but albumin, casts, and even blood may be found in the urine. The most severe and the most characteristic renal complication, and that generally designated as *post-scarlatinal nephritis*, is a diffuse nephritis, with changes in the glomeruli as the

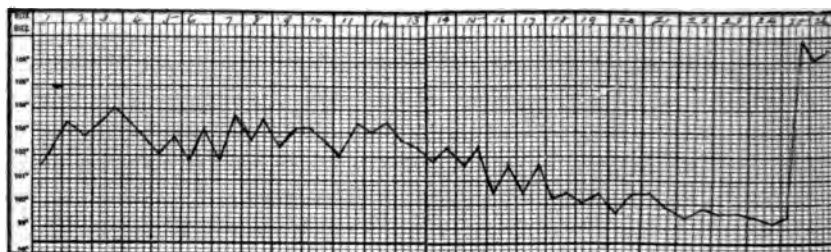


FIG. 185.—SCARLET FEVER OF MODERATE SEVERITY FOLLOWED BY FATAL NEPHRITIS. Early symptoms typical and uncomplicated; twenty-first day vomiting; twenty-fifth day uræmic convulsions; death twenty-sixth day. No dropsy; urine never below 10 ounces in twenty-four hours; girl ten years old.

most striking feature. It usually develops during the third or fourth week of the disease, and may follow mild as well as severe cases (Fig. 185). The onset may be gradual, with dropsy and urinary changes, usually accompanied by a slight rise of temperature; or it may be abrupt, without dropsy but with convulsions, suppression of urine, and very high temperature.

The characteristic urine is of a reddish or smoky colour and scanty. It contains a large amount of albumen, often sufficient to render the urine solid upon boiling. Under the microscope there are seen red blood cells, pus cells, epithelial cells, and casts of every variety. Death may take place from acute uræmia, or the attack may be followed by permanent damage to the kidneys. It is more fully described with the Diseases of the Kidney.

*Joints.*—Acute articular rheumatism may occur coincidently with the development of the scarlatinal rash, and occasionally during convalescence in patients who have a predisposition to that disease. Acute swelling of the joints is sometimes of pyæmic origin. In pyæmic arthritis the large joints are usually involved and the lesions are apt to be multiple. Joint disease may occur as a sequel of scarlet fever, when it is secondary to disease of the bone or to periarticular abscesses opening into the joint.

The foregoing include but a small proportion of the joint complications seen in scarlet fever. The most frequent and most characteristic form of inflammation is *scarlatinal synovitis*, often improperly called *scarlatinal rheumatism*. It occurs in different epidemics with varying frequency. Carslaw (Glasgow), in 533 cases of scarlet fever, met with synovitis in 60 patients. It is seldom seen in children under three years

of age, and is most frequent after five years. It may occur in mild as well as in severe cases. According to Ashby, synovitis develops toward the end of the first or the beginning of the second week. The symptoms are generally mild, and are followed by prompt recovery. Suppuration is rare. Any of the joints may be attacked, but those of the wrist, hand, elbow, or knee are most frequently affected. The symptoms are redness, moderate pain, swelling, which is usually due to synovial distention, and sometimes a slight rise in temperature. The duration is generally but three or four days, and in most cases there is spontaneous recovery. Besides these milder cases there occurs a much more severe form, which may develop later, even during convalescence. It is not very acute, but is accompanied by fever, and both the fever and swelling may continue for many weeks. Recovery may be complete or some joint disability may remain and chronic arthritis may follow.

*Lungs.*—The pulmonary complications of scarlet fever are neither so frequent nor so important as those of measles. Broncho-pneumonia is usually found at autopsy in septic cases where death has occurred later than the third or fourth day, but it is not generally recognisable so early by physical signs.

In septic cases pleuro-pneumonia sometimes occurs early in the disease and at other times late, generally associated with nephritis, but occasionally without it. It is always a serious condition, and not infrequently a direct cause of death. Empyema may follow pleuro-pneumonia or occur with pyæmia or nephritis, but with the latter, simple serous pleurisy is more common. Edema of the lungs occurs chiefly with nephritis, in which it is the most common cause of death.

*Heart.*—Cardiac murmurs are frequent at the height of the disease; in fact they are heard in almost all severe cases. Endocarditis and pericarditis are oftenest seen in septic cases, and with post-scarlatinal nephritis. Endocarditis may be simple or malignant, and may lead to embolism during convalescence. Some degenerative changes in the cardiac muscle are probably present in all the severe cases. Acute dilatation may result, which is sometimes a cause of death.

*Blood.*—In all cases there is a rapidly progressing anæmia that lasts into convalescence. The reduction in the red cells in an average case is about one million. The chief interest, however, attaches to the number and character of the white cells. In mild cases there may be only a moderate increase in their number, usually to from 10,000 to 14,000. It is in cases of moderate severity that the characteristic changes are found. In these there is a decided leucocytosis which appears early, attains its maximum about the fourth day, and gradually declines until the normal is reached, which may not be until the third, fourth, or fifth week. The maximum is usually about 30,000 to 35,000; although it may be as high as 75,000. During the first week the polymorphonuclear



neutrophiles form from 90 to 95 per cent of these cells; the eosinophiles as well as the lymphocytes are diminished. After the fifth or sixth day there is a rapid increase in the eosinophiles which attain their maximum, sometimes 20 per cent of the total leucocytes, between the fourteenth and twenty-first days. After the third week they gradually diminish. Exceptionally there is found in convalescence a relative lymphocytosis, which may be as high as 50 per cent. Complications, nephritis excepted, usually show actual as well as relative increase in the polymorphonuclear neutrophiles. In malignant and rapidly fatal cases there is usually a very small proportion of eosinophiles, and little if any leucocytosis, though exceptionally it may be high.

*Digestive System.*—Functional disturbances are very frequent, and, in fact, are seen in most of the cases, but organic changes are rare. Vomiting is the mode of onset in the majority of cases, but rarely continues throughout the attack. Late in the disease it is a frequent symptom of uræmia. Diarrhœa may be associated with it under both conditions. The tongue is nearly always coated, and clears off in quite a characteristic way, which, with the prominent papillæ, gives rise to the “strawberry” appearance. Catarrhal stomatitis is a very frequent complication, and in many cases of severe membranous angina the same process is seen in the buccal cavity.

*Nervous System.*—Nervous complications and sequelæ are seen less frequently with scarlatina than with most of the infectious diseases of such severity. Convulsions are frequent at the outset, and generally indicate a severe attack, though not invariably so. Occurring late in the disease, they are usually due to uræmia, and may be a cause of death. Meningitis may occur as a complication of otitis, in pyæmic cases, and sometimes with post-scarlatinal nephritis. Paralysis from peripheral neuritis is rarely seen. Hemiplegia sometimes occurs from meningeal hæmorrhage, or from embolism secondary to endocarditis and associated with nephritis. Chorea was noted as a sequel in only three of 533 cases reported by Carslaw. In a report of 187 cases of epilepsy, Wildermuth states that it followed scarlet fever in 12 cases. Insanity has been occasionally observed, the usual form being acute mania, with complete recovery in a few weeks or months.

*Gangrene.*—Cases of symmetrical gangrene after scarlet fever have been reported. The parts generally affected are the buttocks, thighs, and arms, but it may occur almost anywhere. The pathology of these cases is obscure. The process usually begins in several places simultaneously, or in rapid succession, and advances steadily till death occurs.

*Other Infectious Diseases.*—Diphtheria is most frequently seen, and may be present even when there is no distinct membrane.

Scarlatina may also be complicated by measles, varicella, or facial

erysipelas, and occasionally by variola or typhoid fever. The symptoms are often an irregular commingling of those belonging to the two diseases. They may begin simultaneously, or more frequently one develops as the other is subsiding.

**Diagnosis.**—The characteristic symptoms of scarlet fever are the abrupt onset, usually with vomiting, the marked elevation of temperature, the erythematous condition of the throat, the punctate eruption on the hard palate, with the appearance of the rash within twenty-four hours, and later the characteristic appearance of the tongue. The difficulties of diagnosis usually depend upon irregularities in the eruption. The variations are seen in the mildest and in the most severe cases. In the former the rash may be of short duration, often less than a day, and in consequence easily overlooked; or it may be present only upon certain parts of the body instead of being diffuse. In every doubtful case the groins, axillæ, and loins should be closely scrutinised for a punctate eruption. In very severe attacks also the rash may be uncertain. It may appear late or recede after being fully out, or it may be hæmorrhagic or in irregular blotches instead of a uniform blush. In all cases, too much stress should not be placed upon the rash alone.

Until we have some exact means of laboratory diagnosis as in typhoid fever, malaria, and diphtheria, an absolute diagnosis will in certain cases be impossible. Sometimes the diagnosis remains doubtful until the end, although occasionally confirmatory evidence may be obtained even in convalescence. Thus, a patient may desquamate in a manner so typical as to leave no doubt as to the nature of the preceding illness; again, the occurrence of a characteristic sequel, such as nephritis in the third or fourth week, may testify strongly for scarlatina as the primary disease; and, finally, the outbreak of undoubted cases among children who have been in contact with the patient is practically conclusive, always provided other sources of infection can be excluded. Desquamation, however, follows so many other eruptions that when slight or irregular one should not rely upon it as an evidence of scarlet fever, but only upon a typical exfoliation upon the hands and feet. It is a point of some practical importance not to oil the skin of a patient when awaiting desquamation for diagnosis, as this alters very much the characteristic appearances. In some puzzling epidemics the length of the incubation may be of material assistance in the diagnosis; where this is regularly more than a week, one may be pretty sure that he is not dealing with scarlet fever.

Scarlet fever with severe throat symptoms and doubtful eruption can be distinguished from diphtheria only by cultures, which should be made early and repeated if the first result is uncertain. Measles is distinguished from scarlet fever by the length of the invasion, the catarrhal symptoms, and the slowly spreading eruption, but most of

all by Koplik's spots. Much more difficult is it to distinguish between mild scarlatina and rubella. In rubella the important thing is that, although the rash may be well marked, often covering the body, the constitutional symptoms are few or entirely absent. In scarlet fever with an eruption of the same intensity there is almost invariably a considerable elevation of temperature, usually 102° or 103° F., and a bright red throat.

There are so many skin eruptions which may resemble that of scarlet fever, that it is always hazardous to make the diagnosis of this disease from the eruption alone. This is especially true of sporadic cases occurring in infants; there is seen at this age a great variety of eruptions, usually associated with digestive disturbances, which closely simulate a scarlatinal rash; but most of them are of short duration. A scarlatini-form erythema is occasionally seen after diphtheria antitoxine, also in influenza, typhoid fever, pneumonia, and varicella, which may cause them to be mistaken for scarlet fever, or may lead to the conclusion that both diseases are present. The same is the case with the septic erythema occurring in surgical patients. Belladonna, quinine, and occasionally antipyrine, the salicylates and aspirin may produce eruptions more or less closely resembling that of scarlet fever. This is also true of some cases of urticaria and other forms of skin disease. Eruptions resembling scarlet fever may also arise from irritation due to clothing, to heat, to the local application of irritants to the chest, such as camphorated oil, etc. There is little doubt that many of the cases reported as relapsing scarlatina are really examples of recurring erythema, particularly as some of the latter are followed by a desquamation which is very similar to that after scarlatina. In all doubtful conditions great importance is to be attached to the constitutional symptoms.

**Prognosis.**—There is no disease in which it is more difficult to foretell the outcome than in scarlet fever. Cases apparently of the mildest type not infrequently develop serious symptoms and even complications that could not be foreseen. Symptoms indicating a bad prognosis are, very high temperature, especially one which continues to rise for the first three or four days, and severe nervous and throat symptoms. The most common cause of death is the disease itself, the scarlatinal toxæmia. From this cause more than half the deaths occur. Next are the complications, cardiac, pulmonary, renal, otitic, mastoid and cerebral, given in the order of their frequency. The mortality of scarlet fever varies much in different epidemics. In some, nearly all the cases are of a mild type, and the mortality may be as low as 3 or 4 per cent; in others, a severe or malignant type prevails, and it may be as high as 40 per cent. The disease is, as a rule, more fatal in the youngest infants, becoming less so as age advances. This is well shown in two epidemics in the New York Infant Asylum. There were—

|                                   |                                   |
|-----------------------------------|-----------------------------------|
| Under one year . . . . .          | 29 cases; mortality, 55 per cent. |
| From one to two years . . . . .   | 37 " " 22 " "                     |
| From two to three years . . . . . | 28 " " 7 " "                      |
| Over three years . . . . .        | 23 " " 0 " "                      |

In the first epidemic the general mortality was 12.5 per cent; in the second it was 33 per cent in the same class of children.

The following are the mortality records from various European sources:

|  |                                      |
|--|--------------------------------------|
| Ashby, Manchester Hospital . . . . .                     | 681 cases; mortality, 12.2 per cent. |
| Koren, a single epidemic . . . . .                       | 428 " " 14.0 " "                     |
| Bendz, Copenhagen . . . . .                              | 22,036 " " 12.2 " "                  |
| Ollivier, three Paris hospitals for five years . . . . . | 893 " " 14.0 " "                     |
| Fleischmann, five epidemics . . . . .                    | 1,356 " " 10.0 " "                   |

The general mortality of the disease may therefore be assumed to be from 12 to 14 per cent; it is, however, much higher than this among young children, as shown by the following figures:

|                                      |  |
|--------------------------------------|--|
| New York Infant Asylum . . . . .     | 116 cases under 5 years; mortality, 20 per cent. |
| Ashby, Manchester Hospital . . . . . | 259 " " 5 " " 23 " "                             |
| Bendz . . . . .                      | not stated " 5 " " 13 " "                        |
| Heubner . . . . .                    | 136 cases " 7 " " 30 " "                         |
| Fleischmann . . . . .                | not stated " 4 " " 43 " "                        |

Under five years of age the average mortality from scarlet fever is, therefore, between 20 and 30 per cent.

**Prophylaxis.**—Even the mildest cases should be isolated for four weeks, and all cases until desquamation is complete. If complications exist, such as otitis, rhinitis, pharyngitis, empyema, or suppurating glands, the quarantine should be continued until these conditions are cured. Patients should not be allowed to mingle with other children for at least a month after all symptoms have subsided, and should be forbidden to sleep with other children for three months. Children in the house who have not been exposed to the disease should be immediately sent away; and those who have been exposed, separately quarantined for at least a week. After recovery, the patient, before mingling with other children, should have at least two disinfectant baths, the entire body being scrubbed with soap and water and then washed in a solution of carbolic acid (1 to 50) or bichloride (1 to 5,000), and every particle of clothing changed. The hair and the scalp should be thoroughly washed and disinfected.

The nurse should be quarantined with the patient, and should not mingle with other members of the family until a complete change of clothing has been made, and hands and face and hair thoroughly disinfected. The nurse and all others in close contact with a severe case should use frequently an antiseptic gargle and a nasal spray. The room

should be in that part of the house most easily quarantined, usually on the top floor; during the attack it should be stripped of upholstery, hangings, and carpet, and should be freely ventilated and kept as clean as possible. All dust should be removed with damp cloths which should afterward be burned; the floor should occasionally be sprinkled with a bichloride solution (1 to 1,000). The presence in the room of vessels filled with antiseptic fluids is of no practical value. The same may be said of sheets wet in carbolic or other solutions and hung about the room. Carbolic-acid poisoning has been known to result from this practice. After an attack it should be remembered that the room is probably a greater source of danger than the patient. Smooth walls should be wiped with damp cloths wrung out of a bichloride solution (1 to 2,000). The wood-work should be washed in the same solution and the floor scrubbed with it. After a thorough cleaning, while the floor is still wet and walls damp, the apartment should be fumigated with sulphur, or better with formaldehyde. Of the various methods of generating formaldehyde, that of Wilson<sup>1</sup> is probably the cheapest, simplest, and most effective. If fumigation is to be efficient the room must be tightly closed, all cracks being stopped with cotton, and larger openings about doors, windows, and fireplaces sealed by pasting paper over them. Bedding, cushions, pillows, carpets, etc., should be hung over chairs or upon lines strung about the room. Books should be suspended from covers so that the leaves are exposed. After fumigation, the room should remain closed for twelve hours. After a severe case, the walls should be painted or whitewashed, or if papered, the wall-paper should invariably be renewed and the wood-work repainted. Simply airing a room after an attack is of little or no benefit. An instance is on record of a patient contracting the disease in a room in which the windows had been open constantly for three months. The carpets, bedding, hangings, and upholstery can be disinfected only by steam under pressure. Where this is impossible, after a severe case the mattress and pillows should be burned. Bedding, blankets, and other articles should be boiled.

The bedclothes, linen, and clothing removed from the patient during an attack, should be put at once into a solution of carbolic acid (1 to 20), or zinc sulphate four ounces, common salt two ounces, and water one gallon, and afterward boiled in the same solution. Instead of

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<sup>1</sup> For each 1,000 cu. ft. of space there is required 1 lb. of absolutely quick lime, 6 oz. of a 40-per-cent solution of formaldehyde, 2 oz. of a saturated solution of aluminum sulphate. The ingredients may be mixed in a bucket or bowl, which should stand upon wood or in a vessel containing water, as considerable heat is generated. The lime is first moistened with water; then the two solutions previously mixed are poured on and thoroughly mixed with the lime by stirring. The liberation of the formaldehyde gas takes place very rapidly, practically all of it in fifteen or twenty minutes. For a large room several receptacles are better than a single large one.

handkerchiefs, pieces of old muslin, surgeon's gauze, or absorbent cotton should be used for cleansing the nose and mouth of the patient and burned immediately.

The physician in attendance should leave his coat and overcoat in an anteroom, and put on a cap and a long gown or rubber coat, sufficiently large to cover all his clothing. Rubber gloves may be worn as an additional precaution. The gown and cap should always be worn in the sick-room, and boiled or disinfected when the case is finished. For a single visit the overcoat may be worn in the room, but the clothing should be changed before visits to other children are made. After every visit the physician's hands and face should be thoroughly washed with soap and then with a disinfectant solution. A physician in attendance upon scarlatinal patients should not attend obstetric cases or other patients with recent wounds.

Schools are hot-beds for the spread of scarlet fever. The greatest sources of danger are the mild or walking cases in which the disease has not been recognised, and the clothing of patients who have had a severe form of the disease. As a rule, a child should be kept from school six weeks from the beginning of the attack, and the certificate of a physician should be required before readmission, stating not only that the desquamation is complete, but also that the child is suffering from no sequelæ. Other children in the household should not be allowed to attend schools of any kind during the period of active symptoms; they should be kept at home on the average for a month. This precaution is necessary, first, because they might carry the disease from the patient at home; secondly, because otherwise they might themselves attend school while suffering from the disease in a very mild form or during the period of invasion. When the sick child is completely isolated, the danger from the first source is very slight. During severe epidemics it frequently becomes necessary to close all schools.

During desquamation the spread of the disease may be in a measure prevented by the free use of inunctions and warm antiseptic baths. All the excreta from the patient should be disinfected throughout the disease, best by a carbolic solution (1 to 20). If cases of scarlet fever are to be transported, this should be done only in a vehicle which can be easily disinfected. Under all circumstances as few persons as possible should come in contact with the patient.

In general, it is to be remembered that the danger is first from the patient, secondly from the room, and thirdly from the nurse. Special attention should always be given to the complete and immediate isolation of the first case which appears in an institution or community, which should apply to mild as well as severe forms of the disease.

**Treatment.**—There is as yet no specific for scarlet fever. The physician's duty in the average case consists in (1) establishing proper quar-

antine and the carrying out of adequate means of disinfection; (2) the hygienic care of the patient; (3) directing the diet; (4) watching for complications, especially otitis and nephritis. It should be borne in mind that otitis is rarely accompanied by pain or tenderness, and is recognised only by an examination of the ears; also that severe and fatal nephritis may follow mild as well as severe cases.

Mild attacks require no medicine. Children should be kept in bed for at least a week after the fever has subsided, and upon a diet of milk and farinaceous food with plenty of water for a period of three weeks. This is an important matter in the prevention of nephritis. During the height of the eruption, the intense itching of the skin may be allayed by sponging with a bicarbonate of soda solution, or by inunctions with vaseline, or by the free use of rice or talcum powder. Plenty of fresh air should always be secured in the sick-room. As soon as the fever and rash have disappeared, daily warm baths with soap and water should be used, after which the entire body should be anointed with vaseline, with the purpose of facilitating desquamation. In case the skin becomes irritated by this treatment, bran baths may be substituted for soap and water.

The temperature does not usually require interference when it only occasionally rises to 104° or 104.5° F. But if there is hyperpyrexia, or a temperature which ranges from 104° to 105.5° F. or over, antipyretic measures are called for. Hydrotherapy is much safer and more certain than drugs. Sometimes sponging is sufficient, but in the great proportion of cases the pack or bath is required. The use of water in the reduction of temperature is especially indicated in septic cases with typhoid symptoms, and in those with pronounced cerebral symptoms. The temperature of the water employed will depend upon the duration of its application. It is generally better to use prolonged sponging or bathing with tepid water than water at a lower temperature for a shorter period.

The nervous symptoms are frequently better controlled by ice to the head and by cold sponging than by medication. Antipyretic drugs may be relied upon to control restlessness and promote sleep, and in mild cases to effect a moderate reduction in temperature. Phenacetine is usually to be preferred.

As soon as the pulse becomes weak or rapid and irregular, or the first sound of the heart feeble, stimulants should be given, no matter at what stage of the disease. In septic, or malignant cases, or in those accompanied by severe angina, adenitis, or cellulitis, stimulants should be used freely. Digitalis is especially valuable when the pulse is weak and the tension low. It may be given alone or combined with caffeine; one minim of the fluid extract of digitalis, and gr.  $\frac{1}{2}$  of caffeine being the initial doses for a child of five years.

The erythematous sore throat requires no treatment except the use of a bland gargle. If there is a profuse nasal discharge, gentle nasal syringing with a warm saline or boric-acid solution may be used with the hope of preventing infection of the middle ear. The local treatment of the throat is the same as that of other cases of severe angina.

Milder forms of adenitis require no local treatment. When severe, the glands should be covered with ichthyol, and an ice-bag applied continuously. Popticing almost invariably does harm. If an abscess forms, early incision should be practised.

The ears of patients with severe throat symptoms should be examined daily in order that there may be no delay in performing paracentesis should this become necessary. Any rise in temperature should direct attention to the ears. The indications for the operation are the same as in other severe forms of otitis.

The physician should be constantly on the watch for the development of nephritis, not only during the febrile period, but also during convalescence. Repeated examinations of the urine are absolutely necessary. These are much facilitated by having a rack of test tubes and the ordinary reagents for detecting albumin in the sick-room, so that the physician may himself examine daily a fresh specimen of urine. The nurse should be instructed to measure and record accurately the twenty-four hours' urine throughout the attack. The treatment of scarlatinal nephritis has been considered in the chapter devoted to Diseases of the Kidney. Diffuse cellulitis of the neck calls for free, early incision as the only means of preventing extensive sloughing.

Sera prepared by means of several different varieties of streptococci have been produced and extensively used without any uniform or striking success. One has been produced by Moser (Vienna), concerning whose effects there is much more favourable evidence. Escherich, Bokay, and other reliable Continental observers in their reports have declared that its effects are not less striking than those obtained from diphtheria antitoxine. It is not yet available in this country.

During convalescence, the urine should be frequently examined; antiseptic gargles and a nasal spray should be used as long as a purulent discharge from the nose or pharynx continues.

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## CHAPTER II.

### MEASLES.

(*Rubeola, Morbilli.*)

MEASLES is an epidemic contagious disease, more widely prevalent than any other eruptive fever; very few persons reach adult life without



contracting it. One attack usually confers immunity. It is highly contagious even from the beginning of the invasion, and spreads with great rapidity from the patient to all susceptible persons exposed. The infectious agent, however, does not cling so long to clothing or apartments as does that of scarlet fever. Measles has a period of incubation of from eleven to fourteen days; a gradual invasion of three or four days with symptoms of an acute coryza, and a maculo-papular eruption which appears first upon the face and spreads slowly over the body, and which lasts from four to six days. This is followed by a fine bran-like desquamation, which is complete in about a week. The mortality is low, except among infants and delicate children, in whom it may reach 30 or even 40 per cent. In institutions for infants and young children no disease is more to be dreaded than measles, not only on account of its severity, but from the frequency with which, in such subjects, it is complicated by broncho-pneumonia.

**Etiology.**—The essential cause of measles is as yet unknown. It is generally believed to be due to a micro-organism, but, as in the case of scarlatina, all attempts to isolate it have thus far been unsuccessful. The virus is one which possesses remarkable powers of diffusion, but whose viability is much less than that of most of the pathogenic germs which are known. Only a short exposure is required to communicate the disease, and even close proximity to a patient does not seem necessary. One instance has come under my own observation where measles was apparently conveyed by an exposure of half an hour across a hospital ward, a distance of at least fifteen feet.

**Predisposition.**—Very young infants do not so readily contract measles, but all other children are extremely susceptible. The disease broke out in a cottage of the New York Infant Asylum which was occupied by twenty-three children, nearly all of them being under two years old; only four escaped, all these being under five months old. In an epidemic reported by Smith and Dabney, 110 unprotected children, between the ages of eight and eighteen years, were exposed and only two escaped. In the Nursery and Child's Hospital, during an epidemic, there were 62 children over two years of age; five were protected by a previous attack and escaped; of the remaining 57 children, 55 took the disease. There were also in the institution 113 children under two years old; of this number 78 per cent took the disease; but, although a number were exposed, not one child under six months old contracted measles. The age of the persons affected depends much upon the length of time since the last outbreak of the disease. In an epidemic occurring in the Island of Guernsey, where the disease had not prevailed for many years, all ages were affected, the youngest being twelve days old, and the oldest, a man and wife, each aged eighty years. Somer has reported an instance of an eruption of measles appearing in a child twelve hours after birth;

the mother was suffering from the disease at the time. Gautier has collected six additional cases, where measles either existed at the time of birth or developed within a few hours after it.

Except, then, in early infancy, the probabilities are very strong that every child exposed to measles will contract the disease. Occasionally, however, one is seen who seems insusceptible to the poison, no matter how close the exposure.

Epidemics of measles are more frequent and more severe during the winter and spring months. They are least frequent and mildest during the autumn months.

*Incubation.*—In 144 cases,<sup>1</sup> in which the period of incubation could be definitely traced, it was as follows:

|   |          |
|---|----------|
| Incubation of less than nine days . . . . . | 3 cases. |
| “ “ nine or ten days . . . . .              | 22 “     |
| “ “ eleven to fourteen days . . . . .       | 95 “     |
| “ “ fifteen to seventeen days . . . . .     | 19 “     |
| “ “ eighteen to twenty-two days . . . . .   | 5 “      |

Thus in 66 per cent of the cases the incubation was between eleven and fourteen days, and in only one case was it less than a week. The constancy of the incubation period is strikingly shown in some epidemics. Thus in the one reported by Smith and Dabney in an institution in Virginia, exactly eleven days after the rash appeared in the first case, the disease developed in twenty children—no cases having occurred in the interval.

*Duration of the Infective Period.*—This is much shorter than in scarlet fever, and the average duration may be placed at three weeks. Haig-Brown discharged fifty-eight cases on or before the twenty-ninth day of the disease, and in no instance was measles spread by these children. Ransom, however, records one instance in which it was communicated thirty-one days after the appearance of the rash.

Measles is highly contagious from the beginning of the catarrhal symptoms. A case occurred in the Babies' Hospital under my own observation, in which a child conveyed the disease four days before the rash appeared. Ransom reports another precisely similar. An instance has been related to me by Dr. S. W. Lambert, where, of thirteen little girls who were at a children's party, only one escaped measles, the source of infection being a child who showed no rash until the following day; the child who escaped had previously had measles. The period of greatest contagion is still a matter of dispute, the general belief being that it is coincident with the highest temperature, the full eruption, and the most severe catarrhal symptoms.

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<sup>1</sup> About twenty-five of these are taken from my own records; the remainder are mainly isolated cases, scattered through medical literature. The incubation is reckoned from the time of exposure to the beginning of the catarrh.

With the fading of the eruption and the subsidence of the catarrh, the communicability of measles diminishes rapidly. It is relatively feeble during desquamation, and soon after this period it usually ceases altogether. It is generally proportionate to the severity of the catarrhal symptoms, and when these are protracted it is probable that the disease may be communicated for a much longer period than that mentioned.

*Mode of Infection.*—Measles is usually spread by direct contagion, very infrequently through the medium of clothing, furniture, or a third person. Measles rarely if ever clings to apartments for weeks or months, as does scarlet fever. Many instances are on record in which the disease has been carried by a third person; but, after all, this rarely happens, unless the contact both with the sick and the well child is very close and the interval short. It is very seldom that measles is carried by a physician who takes even ordinary precautions. In a case reported by Girom, the clothing of a patient is stated to have conveyed the disease nineteen days after an attack, but this must be regarded as very exceptional.

*Lesions.*—The only constant lesions of measles are those of the skin and the mucous membranes, chiefly of the respiratory tract. According to Neumann, the process in the skin is of an inflammatory character, but is more superficial than in scarlet fever. There is congestion, accompanied by an exudation of round cells about the small blood-vessels, and also about the sweat and sebaceous glands, and the papillæ. To this exudation and the œdema, the swelling of the skin is due. It occurs everywhere, but is especially noticeable upon the face.

The changes in the mucous membranes are quite as much a part of the disease as are those of the skin. There is a catarrhal inflammation affecting the conjunctivæ, nose, pharynx, larynx, trachea, and large bronchi, which varies in intensity with the severity of the attack. In the most severe forms in infants and in young children, this inflammation extends with great uniformity to the small bronchi, and usually to the air vesicles, causing broncho-pneumonia. In severe cases, the lesion in the pharynx and larynx also, instead of being catarrhal, may be membranous; the larynx being much more frequently involved, and the ears much less so, than in scarlet fever. Freeman has described areas of focal necrosis in the liver similar to those found in diphtheria; they were present in four of twelve cases examined. The lesions of the lungs and of other organs will be more fully considered under Complications.

The bacteria which are associated with the lesions of the respiratory tract are the staphylococcus and the streptococcus, separately or together, and either form may be associated with the pneumococcus (see Bacteriology of Broncho-Pneumonia). Measles produces conditions in the mucous membranes of the respiratory tract which are especially favourable for the development of these bacteria. They are present in the

mouth in great numbers; they may cause pneumonia, otitis, and other local inflammations, and the pneumococcus or streptococcus may produce a general septicæmia.

**Symptoms.**—*Invasion.*—As a rule, the invasion of measles is gradual, both the fever and catarrhal symptoms increasing steadily up to the appearance of the eruption. The characteristic symptoms of the invasion are those of a severe coryza—suffusion of the eyes, increased lachrymation, photophobia, sneezing, and a discharge from the nose. The hoarse, hard cough indicates that the catarrhal process has involved the larynx and trachea, as well as the visible mucous membranes. Frequently the patient complains of some soreness of the throat, and on inspection there is seen moderate congestion of the tonsils, fauces, and pharynx. On the hard palate are frequently seen small red spots. Much more characteristic are the minute white spots upon the mucous membrane of the cheeks, known as Koplik's sign (see Diagnosis). The constitutional symptoms are indefinite, and may be met with in almost any disease. There are dulness, headache, pains in the back, and the usual symptoms of malaise; there is rarely vomiting or diarrhœa. Drowsiness is a frequent symptom, and is regarded by the laity as characteristic.

The exceptional cases in which the invasion is abrupt are puzzling. There may be a sudden accession of fever with vomiting, and even convulsions, as in a case lately under my observation. Not infrequently, when the disease prevails epidemically, the invasion is sudden, with high fever and pulmonary symptoms which are so severe as to mask everything else until the rash makes its appearance, the case up to that time being often regarded as one of primary pneumonia or of influenza. The duration of the stage of invasion—i. e., from the beginning of the catarrh until the eruption—in 270 cases of which I have notes, was as follows:

|                         |           |                  |           |
|-------------------------|-----------|------------------|-----------|
| 1 day or less . . . . . | 35 cases. | 6 days . . . . . | 20 cases. |
| 2 days . . . . .        | 47 "      | 7 " . . . . .    | 6 "       |
| 3 " . . . . .           | 64 "      | 8 " . . . . .    | 2 "       |
| 4 " . . . . .           | 64 "      | 9 " . . . . .    | 2 "       |
| 5 " . . . . .           | 29 "      | 10 " . . . . .   | 1 case.   |

From this table it will be seen that the length of the period of invasion varies considerably—more, I think, in infants and very young children (most of these were under three years old) than in those who are older. In the greater number of cases it lasts from two to four days.

**Eruption.**—The rash usually appears on the third, fourth, or fifth day of the disease—in the largest number upon the fourth day. As a rule, it is first seen behind the ears, on the neck, or at the roots of the hair over the forehead. It appears as small, dark-red spots, which are at first few, scattered, and not elevated, resembling flea-bites. In twenty-four hours the macules are much more numerous, and many of them

have become papules. They frequently group themselves in crescentic forms. They are usually separated by areas of normal skin, but where the rash is intense they are frequently coalescent. From the time of its first appearance to the full development of the rash on the face, is usually about thirty-six hours, but may be from one to three days. With a full eruption (Plate XVI) there is considerable swelling of the face, especially about the eyes, and the features are sometimes scarcely recognisable. On the second day of the rash it begins to appear upon the neck beneath the chin, the upper part of the chest and back; on the third day the trunk is covered, and scattered spots are seen upon the extremities. The rash appears last upon the lower extremities, and by the time it is fully out upon them it has usually begun to fade from the face. In mild cases it remains discrete, but in severe ones it is frequently confluent upon the face and upon the extensor surfaces of the extremities. As a rule, it covers the entire body, even the palms and soles.

The eruption fades slowly in the order of its appearance, and there is left behind, in typical cases, a slight brownish staining of the skin which often remains for a week or more. The duration of the rash is from one to six days, the average being four days.

There are many cases in which the rash does not follow the typical course described: (1) Instead of spreading gradually, the entire body may be covered in a few hours. (2) The rash may be hæmorrhagic. This condition was present in about five per cent of my cases. The whole eruption may be hæmorrhagic, or it may be so only upon certain parts—usually the abdomen or extremities. Under such circumstances small petechial spots take the place of the macules—the “black measles” of the older writers. It is in most cases a bad, but by no means a fatal symptom. I have seen it in several cases that were not especially severe. (3) The rash may be very faint, and of short duration, being scarcely elevated at all. (4) It may consist of very minute papules, closely resembling the rash of scarlet fever. It is to be remembered, however, that the irregular eruptions of scarlet fever much more frequently resemble measles than *vice versa*. (5) It may be very scanty, and late in its appearance; particularly in cases of great severity and hyperpyrexia—the so-called malignant cases. (6) Temporary recession of the eruption may occur at any time during the height of the disease, and is usually due to heart failure. A recurrence of the eruption after it has run its usual course is something which I have never seen; although such cases have been reported, I believe them to be very exceptional.

During the first two days of the eruption, the local and constitutional symptoms increase in severity, both usually reaching their maximum at the time of the full development of the rash upon the face. The skin is swollen, and the seat of intense itching and burning. The eyes are very red and sensitive to light, and there is swelling of the conjunctivæ

PLATE XVI.



ERUPTION OF MEASLES.

On the face and trunk the eruption is rather more confluent than is usual: on the upper part of the chest, on the lower part of the abdomen, but especially on the left arm, many hemorrhagic spots are seen. The eruption on the lower extremities and feet is typical in appearance.

with an increase of temperature to 102° or 103°—this, however, the patient seldom notices. The temperature is usually normal in the axilla, but elevated in the rectum and vagina. The pulse is frequent and irregular. There is usually no cough and no coryza. The conjunctivae are red and the eyes are watery and sore, but no symptoms resembling the "conjunctivitis" frequently associated with measles. As the rash fades the temperature gradually returns to normal and the patient improves. The eruption usually subsides and soon the patient is discharged. There is little or no mortality, the fever has seldom exceeded five days.

*Disposition.*—The course of the disease is the first and most important and is first noted in the first or second week after the eruption first appeared. The nature of the disposition is entirely dependent upon the season, never in late periods is a severe fever. In a winter case, however, and after the eruption the fever is more severe than in the summer. It may be accompanied by convulsions. The eruption is usually the most severe in the first or second week. It is most marked in the face and on the neck and chest. There is usually no mortality noted at this time, but in the late periods of the epidemic cases are characterized by marked prostration, the eruption fades and the eyes remain watery and sore, and the patient is discharged. The patient recovers in five or six days.

1. *The course of the disease.*—The first stage is the prodromal or vesicular period, which lasts about five or six days. The eruption is usually first noted in the second or third week. The eruption is usually severe and is most marked in the face and on the neck. The eruption is usually accompanied by convulsions, and the fever is usually severe. The eruption is usually the most severe in the first or second week. It is most marked in the face and on the neck and chest. There is usually no mortality noted at this time, but in the late periods of the epidemic cases are characterized by marked prostration, the eruption fades and the eyes remain watery and sore, and the patient is discharged. The patient recovers in five or six days.

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A temperature curve resembling this was seen in 28 of 173 cases. The rash is often late in appearance; it is faint and altogether irregular; it may recede after the first day and reappear after an interval of one or two days. The catarrhal symptoms are not marked, but the whole force of the disease seems to be expended upon the lungs. The diagnosis of these cases presents great difficulties, and very often it would

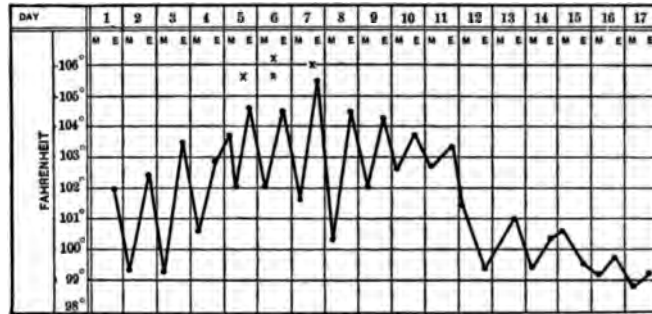


FIG. 189.—MEASLES WITH PROLONGED INVASION. Continuance of high temperature after full eruption due to severe bronchitis and diarrhoea; child two years old.

not be made but for the fact that there are other cases of measles in the family or the institution. This form is usually seen in infants, and it is usually fatal.

In other cases marked by a sudden severe onset, the system seems to be overpowered by the poison of the disease itself. There is profound depression, and hyperpyrexia, and the patient may die from toxæmia with

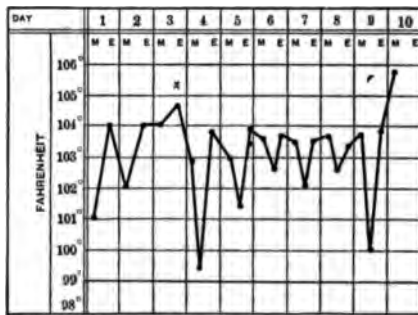


FIG. 190.—FATAL ATTACK OF MEASLES, COMPLICATED BY BRONCHO-PNEUMONIA. Very severe symptoms from the onset; patient eighteen months old; death on tenth day.

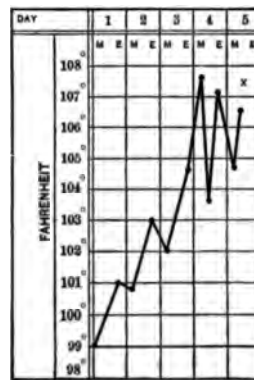


FIG. 191.—FATAL ATTACK OF MEASLES, COMPLICATED BY BRONCHO-PNEUMONIA. Early invasion mild, but rapid development of severe symptoms on fourth day; rash on last day; patient eight months old.

cerebral symptoms before the appearance of the rash or just as it is beginning to show itself. Sometimes the pulmonary symptoms are entirely wanting; at others the rash, if it appears, is hæmorrhagic.



In still another group of cases the onset is not violent, and for the first two days the attack may appear to be of only average severity; but there may then develop, often quite suddenly, pulmonary symptoms of such intensity as to cause death within twenty-four hours. The eruption, if seen at all, is faint and not characteristic (Fig. 191).

A secondary rise in the temperature after it has once fallen to normal was seen in 8 of 173 cases, being due to the development of otitis, ileo-colitis, or pneumonia.

**Complications and Sequelæ.**—The most frequent and most important complication of measles is broncho-pneumonia, and next to this are ileo-colitis, otitis, and membranous laryngitis. Most of the others are infrequent; all complications are relatively rare in children over four years old.

**Lungs.**—The greatest danger in measles arises from pulmonary complications, and the frequency is greatest in children under two years of age. In two epidemics in the Nursery and Child's Hospital, embracing about 300 cases, nearly all in children under three years old, broncho-pneumonia occurred in about 40 per cent of the cases. Of those who had pneumonia, 70 per cent died. Fortunately, such a record as this is never seen outside of asylums or hospitals for young children. Of 2,477 cases, embracing several epidemics of measles among children of all ages, pneumonia occurred in 10 per cent. My own experience in the post-mortem room fully bears out the statement of Henoch, that a certain amount of pneumonia is found in almost every fatal case. Pneumonia is more frequent and its mortality is higher in spring and winter epidemics than in those occurring at other seasons. It may develop at any time from the beginning of invasion until convalescence, but it most frequently begins about the time of full eruption.

Lobar pneumonia, although rare, occasionally occurs as a complication in children over three years old. In some epidemics many of the cases of pneumonia are complicated by severe pleurisy, which adds much to the danger from the disease. This form is frequently followed by empyema. Pneumonia is always to be suspected when the temperature continues high after the full appearance of the rash.

Bronchitis of the large tubes, always accompanied by tracheitis, is seen in every case of measles, possibly excepting a few of the very mildest. This is so constant a feature as hardly to be ranked as a complication. In nearly all of the severe cases the bronchitis extends to the medium-sized and smaller tubes.

**Larynx.**—A mild catarrhal laryngitis accompanies almost every case of measles. Severe catarrhal laryngitis is present in about ten per cent of the cases; it may give symptoms which closely resemble those of membranous laryngitis, and the two are no doubt often confused.

Membranous laryngitis is especially seen in the epidemics of insti-

tutions. As a cause of death in older children it ranks next to pneumonia. When it develops at the height of the disease, it is sometimes due to the streptococcus; but when it develops at a later period, it is usually due to the diphtheria bacillus. The streptococcus inflammation is in most cases associated with similar changes in the pharynx or tonsils, but not always. True diphtheria, occurring as a complication of measles, not infrequently begins in the larynx. The streptococcus inflammation may be as serious in this connection as is true diphtheria, from the probability, which amounts almost to a certainty, of the development of broncho-pneumonia. No complication is more to be dreaded than this. The diagnosis between the true and pseudo-diphtheria may sometimes be made by the time of development, but only with certainty by cultures. I once saw in measles, where no false membrane was present in the rest of the larynx, a necrotic inflammation with almost entire destruction of the vocal cords—a condition which may be compared to that seen in the tonsils or epiglottis in scarlatina.

*Throat.*—A catarrhal angina is part of the disease, and is as characteristic of measles as is the eruption upon the skin. There is acute congestion and swelling of the tonsils, uvula, palate, and pharynx. In a certain proportion of cases, very much less frequently than in scarlatina, the development of membranous patches is seen upon the tonsils and adjacent mucous membranes. These occur in two or three per cent of the cases. They are to be regarded in the same light as similar conditions complicating scarlet fever, with these differences, that in measles there is much greater likelihood of the extension of the disease to the larynx, while extension to the nose and ears is much less probable. True diphtheria, however, may complicate measles, and cases of membranous inflammation of the tonsils or pharynx developing late in measles are usually due to the Klebs-Loeffler bacillus.

Although in most cases the inflammations of the pharynx and tonsils which accompany measles are not serious when they are due to the streptococcus, they are sometimes quite as severe as any that accompany scarlet fever. They may cause death from general sepsis apart from any affection of the larynx.

*Digestive System.*—Gastric disorders are not more common than in other febrile diseases; but diarrhoea is very frequent, and in summer it may be even more serious than the pulmonary complications. All forms of diarrhoea are seen, from that which results from simple indigestion to the severe types of ileo-colitis. This complication is most often seen in children under two years old. The most severe intestinal symptoms are not usually seen at the height of the primary fever; but, beginning at this time, they often increase in severity, and are most marked in the second and third weeks of the disease.

Catarrhal stomatitis is present in almost every case of measles; less

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*Hæmorrhages.*—Associated with the hæmorrhagic type of the eruption, severe and even fatal hæmorrhages may occur from the mucous membranes, and the latter are sometimes seen without the hæmorrhagic eruption.

*Blood.*—There is a leucocytosis of 15,000 to 30,000 beginning soon after infection, even before the invasion, and increasing for four or five days. During the eruption the number of leucocytes falls gradually to normal or below. A marked leucocytosis at this time or later points to a complication, but its absence during eruption does not exclude one. The differential count shows the increase to be in the polymorphonuclear cells.

*Other Infectious Diseases.*—Measles in institutions is often complicated by diphtheria. Scarlet fever or varicella occasionally occurs during measles, though it is rare that the two eruptions are exactly simultaneous. Epidemics of measles and whooping-cough frequently occur together or follow each other. The relation of measles to tuberculosis seems to be particularly close. In some cases general or pulmonary tuberculosis follows directly in the wake of measles, which seems to furnish, especially in the lungs, conditions which are favourable for the development of latent tuberculosis. As a late manifestation the most common one is tuberculosis of the bones, occurring as hip-joint disease, caries of the spine, etc. An attack of measles in a child with tuberculous antecedents should, therefore, always be looked upon with apprehension.

*Diagnosis.*—A sign of the greatest diagnostic value is the buccal eruption. Although it appears that this was described many years ago by Flindt, of Denmark, it is to Koplik, of New York, that the credit belongs of its independent discovery and publication in 1896. It is generally known as "Koplik's sign." The unit of the eruption is a bluish-white speck upon a red ground; only a few of these may be present or the mucous membrane may be fairly peppered with them (Plate XVII). Often they are not seen except by careful search, for which strong sunlight is necessary; artificial light is not satisfactory. The spots are best seen on the inside of the cheeks opposite the molar teeth, and in most cases only there; but they may be present on almost any part of the buccal mucous membrane. Their diagnostic value is due to the fact that they are nearly always present, that they are not found in other diseases, and that they usually appear two or three days before the skin eruption. They generally disappear at the time of full eruption.

I have recently had an opportunity to study the value of this sign in two epidemics of measles at the New York Foundling Hospital. Careful notes were kept in the second epidemic of 187 cases. Koplik's spots were unmistakably present in 169 cases, absent in 8, doubtful in 10. In 78 cases, fever, rash, and Koplik's spots were all present at the first observation. In 54 patients the sign was noted one day before the rash;

in 25, two days before; in 4, three days before; in 3, four days before; and in 2, five days before. In 2 the spots were not seen until after the skin eruption; in one case they were present without any eruption. As this patient had been exposed and had a prolonged fever, it seems fair to regard the case as one of measles. In only one case was the buccal eruption seen before any elevation of temperature.

These facts, amply confirmed by other observations, indicate that Koplik's sign is of value in enabling us to make a diagnosis from one to three days before it is possible by the skin eruption, also in furnishing a new means of distinguishing measles from the other eruptive fevers, as well as from rashes due to drugs, antitoxine, etc.

Other important symptoms are the coryza, the gradual rise in temperature, and the eruption which appears first upon the neck and face, and slowly extends over the body. Cases which present the greatest difficulties in diagnosis are usually the very severe ones and those in infants.

**Prognosis.**—This depends upon the age and previous condition of the patient, the character of the epidemic, and the season of the year. Except in children under three years of age, the deaths from measles are few; but in institutions containing young children, no epidemic disease is so fatal.

The general mortality of the disease is from 4 to 6 per cent; but in epidemics in institutions for young children it has, in my experience, ranged from 15 to 35 per cent. The following table gives the figures of an epidemic in one institution:

|                                     |                                   |
|-------------------------------------|-----------------------------------|
| From six to twelve months . . . . . | 42 cases; mortality, 33 per cent. |
| “ one to two years . . . . .        | 51 “ “ 50 “ “                     |
| “ two to three years . . . . .      | 27 “ “ 30 “ “                     |
| “ three to four years . . . . .     | 20 “ “ 14 “ “                     |
| “ four to five years . . . . .      | 3 “ “ 0 “ “                       |

In any single case the important symptoms for prognosis are the temperature and the character of the eruption. An initial temperature above 103° F., or one which remains high until the eruption appears, is a bad symptom. So also is one which rises after a full eruption, or which does not fall as the rash fades. The following table shows the highest temperature and mortality in 161 hospital cases:

|  |                                 |
|--|---------------------------------|
| Highest temperature not over 102° F. . . . . | 6 cases; mortality, 0 per cent. |
| “ “ 102° to 103.5° F. . . . .                | 14 “ “ 7 “ “                    |
| “ “ 104° “ 104.5° F. . . . .                 | 49 “ “ 16 “ “                   |
| “ “ 105° “ 105.5° F. . . . .                 | 65 “ “ 40 “ “                   |
| “ “ 106° F. or over . . . . .                | 27 “ “ 80 “ “                   |

A favourable eruption is one of a bright colour, covering the body, remaining discrete, and spreading gradually. It is unfavourable for the eruption to appear late, to be very faint, scanty, or hæmorrhagic, or to recede suddenly, as this is usually due to a weak heart.

PLATE XVII.



A



B

THE BUCCAL ERUPTION OF MEASLES (KOPLIK'S SPOTS).

A. This represents the earliest stage; the spots are few, rather large, widely separated, and usually show a distinct areola; the mucous membrane is normal in color.

B. The later appearance and that most frequently seen.

Near the center of the field the spots are closer together, although still remaining individually distinct; the mucous membrane is somewhat congested. At the margin of the field they are fainter and lack the areola, representing a still later period, such as is seen before they disappear altogether, although in some cases they are not more distinct than this at any stage.



Of 51 fatal cases, the cause of death was broncho-pneumonia in 45, ileo-colitis in 4, and membranous laryngitis in 2. More than half the deaths occurred during the second week, the earliest being upon the fifth day of the disease.

The ultimate result of an attack of measles may not be evident for some time. Cases in which the temperature persists for two or three weeks without assignable cause after the disease is apparently over, should be watched with the greatest solicitude. The explanation of this is most frequently to be found in the lungs, although the physical signs are often obscure. The condition may be either subacute pneumonia or pulmonary tuberculosis. Even though the attack of measles may not have been in itself severe, seeds are often sown the full fruits of which are not seen until long afterward. Chronic glandular enlargements which may or may not be tuberculous, chronic bronchitis, chronic laryngitis, subacute or chronic nasal catarrh, hypertrophy of the tonsils, and adenoid growths of the pharynx—all are frequent sequelæ.

**Prophylaxis.**—Measles is often regarded by the laity as so mild a disease that its prevention is thought to be of little importance, and no effort is made to limit its extension. The great probability that every person at some time in his life will have the disease, is no justification of unnecessary exposure. Although in older children measles is usually mild, this is not so in infants, who should be carefully protected from exposure. Special care should also be taken to avoid the exposure of delicate children or those with a strong tendency to pulmonary disease or to tuberculosis. In institutions it is of the utmost importance to secure prompt and complete isolation of the first case which appears.

The disease being usually spread by the patient and rarely from apartments, it follows that while early isolation is more important, there is not required the same thorough cleansing and disinfection which should follow every case of scarlet fever. In an institution, the ward or cottage from which a case has been removed should be quarantined for at least sixteen days after the appearance of the last case, and absolute security can not be said to exist until the end of three weeks. The same rule should be applied in private families where children who have been exposed should be quarantined apart from the patient, but not sent away. Under ordinary circumstances the quarantine of a case of measles should last three weeks from the beginning of invasion. It should be continued longer if there is pneumonia, otitis, or a nasal discharge.

Thorough cleansing and disinfection of the sick-room should be done before it is again occupied by children, and it should remain vacant at least two weeks. Children should be kept from all schools while the disease is in their homes, chiefly because they are otherwise liable to spread the disease while suffering from the early symptoms of invasion.



**Treatment.**—Measles is a self-limited disease, and there are no known measures by which it can be aborted, its course shortened, or its severity lessened. The indications are therefore to treat serious symptoms as they arise, and, as far as possible, to prevent complications, which are the principal cause of death.

The sick-room should be darkened, as the eyes are very sensitive to light. Every child with measles should be put to bed and kept there with light covering during the entire febrile period. There can be no possible advantage in causing a child to swelter by thick covering, under the delusion that the disease may be modified thereby. The food should be light, fluid, and given at regular intervals. If the conjunctivitis is severe, iced cloths should be applied to the eyes, which should be kept clean by the frequent use of a saturated solution of boric acid, the lids being prevented from adhering by the application of vaseline or simple ointment. The intense itching and burning of the skin may be relieved by inunctions of plain or carbolised vaseline, or by bathing with a solution of bicarbonate of soda. The cough, when distressing, may be allayed by small doses of opium, either in the form of codeine or the brown mixture. The restlessness, headache, and the general discomfort which accompany the height of the fever may be relieved by an occasional dose of phenacetine or antipyrine. As soon as the rash has subsided, a daily warm bath should be given, followed by inunctions to facilitate desquamation and prevent the dissemination of the fine scales.

The important indications to be met in the severe cases are very high temperature, cardiac depression, and nervous symptoms—dulness, stupor, sometimes coma, or convulsions. In some of the cases there is in addition dyspnoea and cyanosis, showing severe acute pulmonary congestion. For the nervous symptoms and high temperature, nothing is so reliable as the cold bath or pack and the nearly continuous use of ice to the head. I do not think there is any evidence that the use of cold increases the liability to pneumonia; but cold extremities, feeble pulse, and cyanosis, when associated with high temperature, call for the hot mustard bath, although ice should still be applied to the head. The indications for stimulants and the methods of using them are the same as in broncho-pneumonia, which is usually present in cases requiring them.

To diminish the chances of pneumonia, it is necessary that every patient should be kept in bed during the attack, and care exercised to avoid exposure. But still more important is it in hospitals and institutions where most of the cases of pneumonia occur, to allow the patients plenty of air space, never crowding them together in small wards. If possible, cases complicated by pneumonia should be separated from simple cases. The pneumococcus and the streptococcus are found in the mouth in such numbers that systematic disinfection of the mouth may prove of some value.

The danger of diphtheria as a complication may be greatly lessened if during epidemics of measles in institutions every case receives an immunising dose of diphtheria antitoxine.

The bronchitis and broncho-pneumonia of measles should be managed as when they occur as primary diseases, since the coexistence of measles furnishes no new indications. The same is true of the diarrhœa, conjunctivitis, otitis, membranous laryngitis, pharyngitis, and tonsillitis. Should cultures show the presence of the diphtheria bacillus, the case should be treated like one of diphtheria.

During convalescence the eyes should be used very carefully for at least several weeks. Should the cough and slight fever persist, with or without physical signs in the chest, the patient should, if possible, be sent away to a warm, dry, elevated district, as the development of tuberculosis is always to be feared. Cod-liver oil should be given continuously throughout the succeeding cool season, and iron, wine, and other tonics according to indications. The cough itself should be treated as when it follows an ordinary bronchitis, creosote being more generally useful than any other drug.

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### CHAPTER III.

#### RUBELLA.

(*German Measles; Rötheln.*)

RUBELLA is a contagious eruptive fever which is rarely seen except when prevailing epidemically. It is characterised by a short invasion, with mild, indefinite symptoms, usually lasting but a few hours, and by an eruption which is generally well marked but of variable appearance. The constitutional symptoms are very mild, and the disease rarely proves fatal, not often being even serious. For a long time rubella was confounded with measles and scarlet fever, as the eruption sometimes resembles one and sometimes the other disease. Its identity is now fully established, and, as Strümpell well says, its existence is doubted only by those who have never seen it.

Rubella is a contagious, eruptive fever, and not a simple affection of the skin; it prevails independently either of measles or of scarlet fever; its incubation, eruption, invasion, and symptoms differ materially from those of both these diseases; it attacks indiscriminately and with equal severity those who have had measles and scarlet fever and those who have not, nor does it protect in any degree against either of them; it never produces anything but rubella in those exposed to its contagion; it occurs but once in the same individual.

**Etiology.**—Rubella is beyond question contagious, but is decidedly less so than either measles or scarlet fever; so that some observers

have doubted its contagion altogether. It can be communicated at any time during its course, but is especially contagious during the early stage. Epidemics usually prevail in the winter or spring. As in the other eruptive fevers, a striking immunity is seen in infants under six months old; but, with this exception, all ages are liable to the disease.

The incubation of rubella varies considerably; the usual period is from fourteen to twenty-one days, although the limits are from ten to twenty-two days.

**Symptoms.**—*Invasion.*—This is rarely more than half a day, and in many cases no prodromata whatever are noticed, the rash being the first thing to attract attention. In a few cases there are mild catarrhal symptoms, with general malaise and slight fever. At other times there may be vomiting, convulsions, delirium, epistaxis, rigors, headache, or dizziness; but all are to be regarded as very exceptional.

*Eruption.*—Frequently a child wakes in the morning covered with the rash, no symptoms having been previously noticed. It generally appears first upon the face, and spreads rapidly to the whole body, the lower extremities being last covered. Less than a day is usually required for its full development. Exceptionally the eruption comes first upon the chest and back, and sometimes nearly the whole body is covered almost at once. The rash is occasionally observed in the roof of the mouth before it is visible on the face. In a considerable number of cases the entire body is not covered; but the rash is more constantly seen upon the face than upon any other part of the body.

Its character is subject to considerable variation. The eruption is most frequently composed of very small maculo-papules; they are of a pale-red colour, and vary in size from a pin's head to a pea. The spots are usually discrete, but may cover the greater part of the body where it is seen. On the face it is frequently confluent, and often appears here as large, irregular blotches of a red colour. From this description the rash will be seen to resemble that of measles more than that of any other disease. Very often, however, there is a fairly uniform red blush which bears a close resemblance to the rash of scarlet fever; but even in such cases there will nearly always be found upon some part of the body, usually the wrists, fingers, or forehead, some typical maculo-papules. Between these two extremes all variations are seen. The colour of the eruption is sometimes dark red, and rarely it has been noted to be hæmorrhagic. The degree of elevation above the surface is also variable; sometimes this is so marked as to give to the skin a "shotty" feel, while in others the elevation is scarcely perceptible. The duration of the eruption is usually three days. Occasionally it lasts only two days, and it may last but one; it is rare for it to remain as long as four days. It fades in the order of its appearance, and more rapidly than the eruption of

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measles. A slight brown pigmentation of the skin sometimes remains for a few days after the rash.

The highest temperature is coincident with the full eruption; this does not usually exceed 101°, and often it is only 100° F. As a rule, the temperature continues but two days, falling as the eruption fades. Very often the fall to normal is abrupt. Rarely more severe cases are seen in which the fever lasts for two or three days, being 101° or 102° F. during the invasion, and rising to 103° F. or more during the full eruption. The other symptoms are in most cases even less marked than the fever. Occasionally catarrhal symptoms resembling a mild attack of measles are present, or a sore throat suggesting mild scarlet fever; but more frequently all these symptoms are absent. The eruption is usually out of all proportion to the other signs of disease.

Swelling of the post-cervical glands is one of the most constant features of rubella. In most epidemics it is seen in nearly all cases; but as a symptom for differential diagnosis it is not of great importance, as it is not uncommon in measles and scarlet fever. The glandular swelling is most marked at the height of the disease; it is never very great, and subsides slowly without suppuration. Vomiting and diarrhœa are rare. Swelling and itching of the skin are usually present and sometimes marked. There is no leucocytosis in this disease.

Forchheimer has described an eruption on the mucous membrane of the throat, or "enanthem," which he believes to be characteristic. It consists of minute, bright, rosy-red points, seen on the uvula and soft palate, rarely on the hard palate. It is present only in the first twenty-four hours.

*Desquamation.*—This is exceedingly variable. It is sometimes entirely wanting; writers who have observed some fairly typical epidemics have stated that it did not occur. In most cases, however, some desquamation is present, though it may be so slight as to be discovered only by a close examination. It is usually in the form of fine scales over the body and extremities. In a few cases it is more pronounced, and may be in larger flakes or patches.

**Prognosis.**—There are few diseases so free from danger as rubella. Complications and sequelæ are very seldom seen, and when present are usually of the mildest character.

**Diagnosis.**—The principal interest attaching to rubella is in its diagnosis. This is a matter of extreme difficulty, and often it is an impossibility. The characteristic thing about the disease is a well-marked eruption with very few other symptoms. Cases so closely resemble mild scarlet fever that the differentiation by symptoms may be impossible; it must be made by the circumstances under which it occurs, especially a prevailing epidemic. Scarlet fever with a low temperature and abundant rash should always be regarded with suspicion; also an abundant rash

with little or no desquamation. The longer period of incubation in rubella may be of assistance. Koplik's spots furnish a valuable means of distinguishing measles from rubella. These difficulties in diagnosis can be appreciated only by one who has seen epidemics of measles and scarlet fever in institutions, and has watched the mild course of undoubted cases of these diseases which have there occurred.

It is always hazardous to make the diagnosis of rubella unless the disease is prevailing epidemically. Sporadic cases in which this diagnosis is made are, I believe, almost invariably instances of mild measles or scarlet fever. The first cases of rubella in an epidemic are usually overlooked. The continued absence in succeeding cases of the characteristic symptoms and complications of measles or scarlet fever should suggest to the physician that he is probably dealing with rubella.

**Treatment.**—None whatever is required for the disease excepting isolation, which should be complete until the diagnosis is positively determined; after this it is hardly necessary. The individual symptoms and complications are to be treated as they arise.

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## CHAPTER IV.

### *VARICELLA.*

(*Chicken-pox.*)

VARICELLA is an acute, contagious disease, characterised by a cutaneous eruption of papules and vesicles and by mild constitutional symptoms, serious complications and sequelæ being very rare. Although long confounded with varioloid, its existence as a distinct disease has been generally admitted for many years.

**Etiology.**—It is well established that the contagium of the disease is contained in the vesicles, as it may be communicated by inoculation with their contents. The specific poison, however, has not yet been isolated. Varicella is contracted by exposure to another case or through the medium of a third person. It affects children of all ages, one attack being as a rule protective. It is very contagious, resembling measles in this respect. The period of incubation is quite uniformly from fourteen to sixteen days.

**Symptoms.**—Slight fever and general indisposition may be noticed for twenty-four hours before the appearance of the eruption, but in most cases the eruption is the first symptom. It usually appears first upon the face or trunk, as small, red, widely scattered papules. The papules in most cases come in crops, new ones continuing to appear for three or four days, even upon the same part of the body. The earlier ones have generally begun to dry up by the time the later ones appear, so that all

stages of the eruption may be present at one time in the same region, this being one of the diagnostic features. The papules are at first very small, but gradually increase in size, and are surrounded by an areola from one-fourth to half an inch in width. Many of them go no further than this stage, but the majority become vesicular. The vesicles are usually flat, and vary a good deal in size—the largest being about one-fourth of an inch in diameter. The process of drying up generally begins at the centre, which causes a slight depression, giving the vesicle a somewhat umbilicated appearance. The areola is most distinct at the time of the fully formed vesicle, and fades as the latter dries. Crusts now form, which fall off in from five to twenty days, depending upon the depth to which the skin has been involved. In the majority of cases no mark is left, but after the most severe attacks, when the true skin has been involved, scars remain, and occasionally there is quite deep pitting. Such marks are few in number, and are most likely to occur upon the face.

Sometimes, especially upon hands and feet, the vesicle appears without having been preceded by a papule; often there is no areola, and the vesicle resembles a drop of water upon healthy skin. In most cases pustules are not seen, but they may develop in consequence of irritation or infection, the result of scratching, or in children who are poorly nourished. Under these circumstances deeper ulceration may occur, lasting for weeks. In rare cases there may be a necrotic inflammation about the site of the pock, a condition to which is sometimes given the name *varicella gangrenosa*. It is not peculiar to varicella, and is described elsewhere under the head of Gangrenous Dermatitis.

The pocks are usually most abundant over the back and shoulders. In mild cases only twenty or thirty may be found upon the entire body, but in severe cases the skin in certain regions may be nearly covered. The eruption is never confluent. The pocks are usually seen on the hairy scalp, and often on the mucous membrane of the mouth or pharynx—a point of some diagnostic value. In the latter situation the appearance is first as a tiny vesicle, and later as a superficial ulcer resembling that of herpetic stomatitis. Marfan and Halle have described cases of varicella of the larynx. Croupy symptoms were present, and in one case which proved fatal from pneumonia a tiny ulcer was found on the vocal cords.

The temperature is highest when the eruption is most rapidly appearing, this usually being the second or third day. In an average case it reaches only 101° or 102° F., and lasts but two days; in severe cases it may rise to 104° or 105° F., and lasts for four or five days. It falls gradually to normal as the rash fades. The other symptoms are mild and not characteristic.

**Complications.**—The most important complication is erysipelas, which develops about the pocks, particularly when they are deep and at-

tended with some ulceration. I have known of three fatal cases from this cause. Adenitis, either simple or suppurative, and abscesses in the cellular tissue, are occasionally seen. Nephritis is very infrequent, but a number of cases are recorded. It may occur at the height of the disease, but more often at a later period, like the nephritis of scarlet fever. Varicella is quite frequently complicated by other infectious diseases. In the New York Infant Asylum epidemics of varicella and scarlet fever at one time occurred together, and in at least a dozen children both diseases were seen at the same time.

**Diagnosis.**—The diagnosis of varicella is usually easy, provided the following points are kept in mind: first, that the eruption comes out slowly and in crops, so that papules, vesicles, and crusts may be seen upon the skin in close proximity; secondly, that the umbilication is due only to the mode of drying up of the vesicle, which begins at the centre; thirdly, the appearance of the pocks upon the mucous membranes, and the history of exposure. It is distinguished from urticaria and other forms of skin disease by the presence of fever.

**Treatment.**—Although it is usually a trivial disease, isolation of cases of varicella should be enforced in schools and in institutions containing many infants. In the home, unless the other children are delicate or in poor condition, quarantine is unnecessary. The disease may probably be conveyed as long as the crusts are present, hence isolation should be maintained until they have fallen off. In most cases constitutional symptoms of the disease are so mild as to require no treatment.

Locally, the itching, when annoying, may be allayed by sponging with a solution of bicarbonate of soda, a one-per-cent solution of carbolic acid or the use of carbolised vaseline. When the crusts have formed, this ointment or vaseline containing two per cent ichthyol should be applied. Care is necessary to keep the skin clean, and, in the case of infants, to prevent scratching. In severe cases the urine should invariably be examined.

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## CHAPTER V.

### VACCINIA—VACCINATION.

VACCINIA (cowpox) is a febrile disease induced in man by inoculation with the virus obtained either directly from the cow (bovine virus) or from a person who has been inoculated (humanised virus). The disease is not contagious in the ordinary sense of the term, but is communicated by inoculation either accidental or intentional.

The nature of the protection against smallpox which vaccination affords is even now but imperfectly understood. The fact, however, re-

mains one of the best attested in medical history. Its effect when systematically practised is graphically shown in the accompanying chart (Fig. 192). It is the imperative duty of the physician to see to it that every young infant is vaccinated.

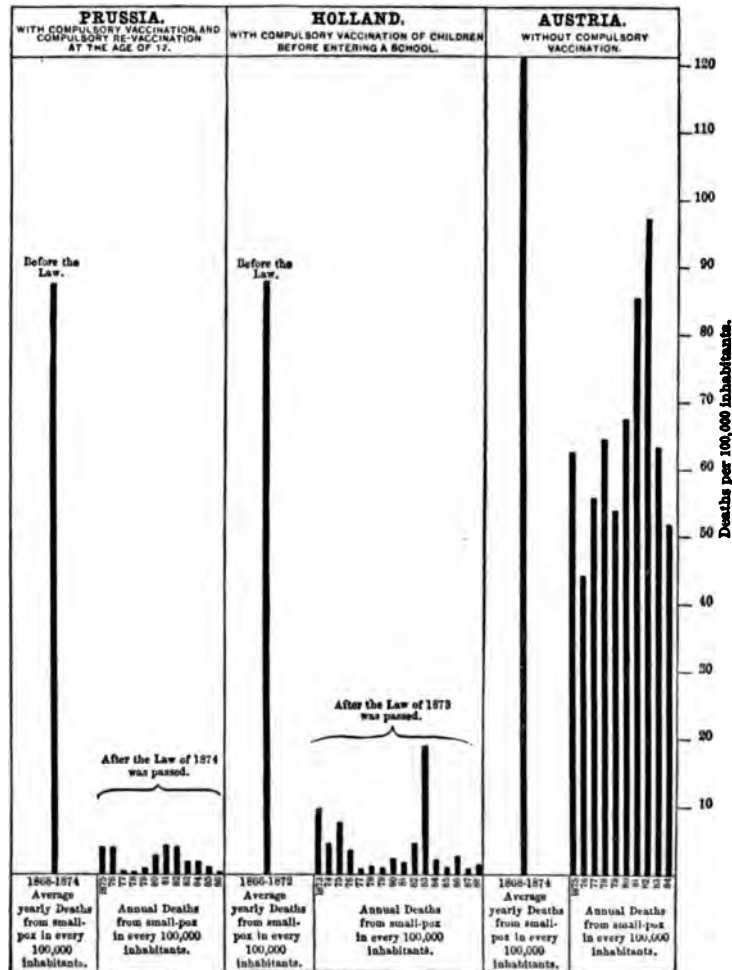


FIG. 192.—TABLE SHOWING THE PROTECTIVE POWER OF VACCINATION. (Carsten.)

**Re-vaccination.**—Regarding the duration of the protective power of a single vaccination, positive statements are impossible. Nearly all writers are agreed that vaccination should be done in infancy, again at puberty, and a third time at about the age of twenty or twenty-five. Many also insist upon re-vaccination at about the seventh year. It is a safe rule when smallpox is prevalent to vaccinate every person who has not been successfully vaccinated within five years.



**Choice of Lymph.**—The substitution of bovine for humanised virus is now well-nigh universal. It has precluded the possibility of transmitting syphilis and greatly lessened the chances of other forms of infection. A further advance has lately been made by the introduction of "glycerinated" lymph. As now prepared, the lymph is taken from the calves under the most rigid aseptic precautions and emulsified with glycerin. The few saprophytic bacteria present soon die, so that when properly prepared the glycerinated virus is practically sterile. It should not be distributed until it has been carefully tested for pathogenic organisms of all kinds, particularly the tetanus bacillus. It is preserved and distributed in capillary tubes hermetically sealed; these are much safer than quills or ivory points, which may easily become contaminated by handling. After the lymph has been taken, the calves are killed in order to make certain that they were free from disease. The practical advantages of glycerinated lymph are so great that it has been officially adopted by the Governments of the United States, Great Britain, Germany, and many other countries.

**Time for Vaccinating.**—In selecting a time for vaccination, the child's age and general health must be taken into consideration. It is pretty well established that the constitutional disturbance is much less in infancy than in later childhood, and less in very young infants (under one month) than in those of five or six months. A good rule for general practice is to vaccinate every healthy infant as soon as its nutrition is established, this being in most cases during the first three months of life. In delicate infants or in those whose nutrition is a matter of great difficulty, those who are syphilitic, those suffering from eczema or any other form of active skin disease, vaccination should be deferred until the child is in good condition, unless it is likely to be exposed to smallpox. As a rule, vaccination should be avoided during dentition.

**Methods of Vaccinating.**—In my experience it is better to vaccinate in one place rather than to make two or three inoculations. If more than one is made they should be at least an inch apart. Either the leg or the arm may be chosen; in young infants it is usually easier to protect the vaccine sore upon the leg than upon the arm; in children old enough to run about, the arm is to be preferred, as being more easily kept at rest. The point selected for inoculation should be either the outer aspect of the left calf, about the junction of the middle with the upper third of the leg, or, if the arm is chosen, the insertion of the left deltoid. The skin should be washed with soap and water, dried, and then washed with alcohol.

The New York Health Department supplies with each tube of lymph, a needle, a bit of rubber tubing, and a sterilised tooth-pick with one flat end. The needle should be sterilised in an alcohol flame, and a single scratch made not more than one-eighth of an inch long, just deep enough to draw blood. The ends of the capillary tube are broken off, one end

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inserted in the rubber tube, and the lymph blown out of the tube upon the broad end of the tooth-pick, then applied to the scratched surface and rubbed in for a full minute. The wound should not be covered until dry; this usually requires from fifteen to twenty minutes. It may then be covered with a sterilised bandage. If thoroughly dried no dressing is necessary. The limb should not be washed for twenty-four hours.

**The Normal Course of Vaccinia.**—The course of a proper vaccination-pock is quite uniform, and one which does not follow this course should not be considered protective. The wound heals and nothing is noticed until the third or fourth day, when a red papule makes its appearance. Usually in twenty-four hours more a small vesicle appears which enlarges until the sixth or seventh day, reaching its full development about the ninth day. Its shape and size depend somewhat upon the scarification (Figs. 193–197). The vesicle is usually from one-fourth to one-half inch in diameter; it is of a pearly gray colour and has a depressed centre. During the next two days an areola forms about the vesicle extending from it a variable distance, usually one or two inches into the healthy skin. Its size depends upon the intensity of the infection. This areola is normally of a bright red colour and accompanied by some induration. It is generally at its height about the ninth day. The vesicle usually dries down to a firm, dark crust which remains from one to three weeks and falls off, leaving a bluish scar which fades to white, becoming somewhat honey-combed. When the process is at its height some constitutional disturbance is usually present; there may be loss of appetite, fretfulness, and general indisposition, and the temperature is usually elevated from one to three degrees. The lymph nodes in the groin or axilla may be tender and swollen. These symptoms generally last for three or four days.

If in a young infant the first inoculation is unsuccessful, at least three trials should be made with good virus, and in the event of further failure, after a year vaccination should be repeated. A failure to inoculate does not mean insusceptibility to smallpox, as is often popularly believed, but most frequently arises from the fact that the virus is inert. I have known one case in which the seventh, and another in which the thirteenth, inoculation was successful after previous failures; occasionally there are seen children who can not be inoculated at all.

Constitutional symptoms, as previously stated, may be absent in very young infants; but in others there is quite constantly present a fever which runs a fairly regular course. It usually begins on the fourth or fifth day, is remittent in type, and rises gradually, reaching its highest point with the full development of the vesicle. At this time even without complications it may touch 104° or 105° F. The duration of the fever in cases running the usual course is four or five days. Accompany-



FIG. 193. Fifth day.



FIG. 194. Seventh day.



FIG. 195. Ninth day.



FIG. 196. Eleventh day.



FIG. 197. Tenth day.

FIGS. 193-197. -VACCINE VESICLES. (Two-thirds natural size.)

FIGS. 193, 194, 195, and 196 show typical appearance of vesicle at the different stages when a very small scarification is made.

FIG. 197 shows the effect of a larger scarification with a more intense areola. The amount of inflammation is excessive but not unusual.

ing the fever there may be anorexia, restlessness, loss of sleep, slight indigestion, and other symptoms of a general indisposition.

Both the local and the general symptoms are sometimes more severe. This may depend upon the susceptibility of the child, even though the lymph is pure and the vaccination properly done. The original vesicle may be much larger than usual, and small secondary vesicles may form in the neighbourhood. In very rare instances a generalised eruption of true vaccine vesicles occurs with fever and other general symptoms of corresponding severity (Fig. 198). Single vesicles may be produced on distant parts of the body as a result of auto-inoculation, usually by scratching. Where eczema of the face is present, inoculation is not infrequently carried thither. Most of the very sore arms and legs, however, are due to infection from pyogenic bacteria contained in the lymph, or to their accidental introduction at the time of vaccination or subsequently. In the milder cases, the swelling and other evidences of local inflammation are more marked than in a normal vaccination; a drop or two of pus forms



FIG. 198.—GENERALISED VACCINIA.  
Boy eight years old.

beneath the scab, and when the latter comes away an excavation is left which heals in two or three weeks. Or, the inflammation may extend more deeply into the connective tissue, to be followed by more extensive suppuration or sloughing, leaving an ugly ulcer an inch or more in diameter which slowly fills by granulation in from five to eight weeks. Sometimes the period of incubation is unduly prolonged, so that the vesicle does not form until the twelfth or fourteenth day, although its subsequent course may be normal. In other cases, the incubation is shorter than usual, and the vesicle may appear as early as the third or fourth day.

Much has been written about the so-called "raspberry excrescence" which not very infrequently takes the place of a proper vesicle. It is of a dark red colour, elevated, smooth or slightly granular, not sensitive, having no areola and no constitutional symptoms. It generally persists for two or three weeks, and slowly disappears, leaving no scar. It is usually the result of virus of feeble activity, and if it gives any protection it is very slight. Such cases should always be re-vaccinated, and in my experience re-vaccination is usually successful.

**Complications and Sequelæ.**—Post-vaccine eruptions are many and of great variety. The most frequent is a general roseola, usually occurring at the height of the local process. Other eruptions seen are urticaria, and, rarely, purpura. Complications are chiefly from accidental infection. Syphilis and tuberculosis are excluded by the modern method of procuring the lymph. Tetanus can result only from carelessness or neglect of suitable precautions in preparing the lymph: proper legal restrictions regarding its production should make this impossible. The most common form of local infection is cellulitis, which may terminate in suppuration or sloughing at the site of vaccination, and sometimes may cause suppuration of the neighbouring lymph nodes. Erysipelas may develop at any time before the skin is entirely healed; it is usually due to neglect of proper precautions in the care of the vaccine sore.

The mortality of vaccination is stated by Voigt, from careful statistics drawn from German sources, to have been 35 in 2,275,000 cases, including both primary and secondary vaccinations. Of the deaths, 19 were due to erysipelas, 8 to gangrene, 2 to cellulitis, 3 to "blood poisoning," and 3 to other causes. The occurrence of tetanus after vaccinia has already been mentioned. With proper precautions in preparing lymph it will not occur. In fact, nearly all the deaths are from causes which are preventable.

**Treatment.**—The whole purpose of treatment is to prevent infection. The first essentials are a clean limb, pure virus, and a sterile needle: the next, to allow thorough drying of the wound before the clothing touches it. After this nothing is necessary until the vesicle forms. Then the important thing is to prevent scratching and the irritation by the clothing. All vaccine shields are objectionable. For an infant nothing is better than the sterilised gauze bandage, which can be kept in place by sewing to the stocking or sleeve of the shirt. Any constriction of the limb is injurious. For older children the simplest dressing is a pad of sterile gauze fastened to the limb by two pieces of adhesive plaster. Should the vesicle rupture and discharge serum, it should be kept clean and dry by dusting daily with boric acid. When the local symptoms are at all severe the limb should be kept at rest. An infected vaccination wound, like any other infected wound, requires careful surgical treatment: disastrous results often follow the use of poultices and other applications much in vogue in domestic practice.

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## CHAPTER VI.

### *PERTUSSIS.*

(*Whooping-Cough.*)

PERTUSSIS is a contagious disease which prevails epidemically and in most large cities endemically. Although it may affect persons of any

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age, it is generally seen in young children, and as a rule it occurs but once in the same individual. While in later childhood pertussis may be ranked as one of the milder infectious diseases, in infancy it is one of the most fatal. Its principal complications are broncho-pneumonia and convulsions. Pertussis is characterised by catarrhal and nervous symptoms. The catarrh affects the mucous membrane of the respiratory tract, and is probably due to a specific form of infection. It is accompanied by a hyperæsthetic condition of this mucous membrane. The most prominent nervous manifestation is a peculiar spasmodic cough which occurs in paroxysms, and from which the disease takes its name. The cough is no doubt of reflex origin, from an irritation which has been located by different writers in various parts of the respiratory tract. In addition to these conditions, there is present in pertussis a marked irritability of the nervous system, which in infancy often shows itself by convulsions.

**Etiology.**—Everything that is known of pertussis suggests a micro-organism as its cause. Present evidence, moreover, points strongly to a bacillus first described by Bordet, although this lacks the final proof of the production of the disease by inoculation. Bordet's bacillus is a small Gram-negative organism which in many points resembles the influenza bacillus. It is cultivated with difficulty, but grows best on potato-blood-agar. Subcultures do not require hæmoglobin for their growth. It is difficult to obtain the organism from the respiratory secretion unless the plug of bronchial mucus brought up after a paroxysm of coughing is secured, as it develops chiefly deep in the respiratory tract. It is found only in the early stage of pertussis, rarely longer than a week after the whoop develops. The influenza bacillus is very frequently associated with it.

Proximity to a patient seems all that is required to communicate the disease, and even close proximity is not necessary. There seems to be no doubt that the disease may be contracted in the open air.

**Predisposition.**—Fully one-half the cases of pertussis occur during the first two years of life. The following are the statistics of Szabo (Buda-Pesth), showing the ages at which the disease was met with in 4,591 cases, comprising the records of one clinic for thirty-four years:

|                              |              |                               |            |
|------------------------------|--------------|-------------------------------|------------|
| Under one year . . . . .     | 1,028 cases. | Three to four years . . . . . | 904 cases. |
| One to two years . . . . .   | 1,008 "      | Four to seven years . . . . . | 803 "      |
| Two to three years . . . . . | 659 "        | Over seven years . . . . .    | 189 "      |

Pertussis thus shows a stronger tendency to affect young infants than does any other contagious disease. A number of cases are on record in which it has occurred during the first month, and one has recently come to my notice where a child twelve days old was attacked, whose mother was suffering from the disease. The disease is nearly twice as frequent in the winter and spring as in the summer and autumn. Epidemics of pertussis often occur at the same time with or follow those of measles.

be symptoms of a mild grade of catarrhal inflammation of the nose, pharynx, and larynx, and often there is a slight elevation of temperature.

*The Spasmodic Stage.*—In a typical paroxysm of average severity the child, who can usually foretell it, will often run for support to the lap of the mother or the nurse, or seize a chair with both hands. There now occurs a series of explosive coughs, from ten to twenty in number, coming in such rapid succession that the child can not get his breath between them; the face becomes of a deep red or purple colour, sometimes almost black; the veins of the face and scalp stand out prominently; the eyes are suffused, and seem almost to start from their sockets; there follows a long-drawn inspiration through the narrowed glottis, producing the crowing sound known as the whoop; and then another succession of rapid coughs follows and another whoop. In a single severe paroxysm, which lasts two or three minutes, the child may whoop half a dozen times; with the final paroxysm a mass of tenacious mucus is usually brought up. In a young child vomiting is almost certain to follow, if food has been recently taken. Epistaxis sometimes occurs with nearly every severe paroxysm, but in most cases the bleeding is slight. After a severe attack the child is at times so exhausted as to be hardly able to stand. There is profuse perspiration; his mind is confused, and he may be completely dazed. In infants the attack may result in a degree of asphyxia requiring artificial respiration. Those old enough to describe their sensations tell of a sense of impending suffocation, the suffering from which is almost indescribable.

The number of severe paroxysms or "kinks" in twenty-four hours varies, according to the severity of the case, from half a dozen to forty or fifty. There are always many more of a milder form. Paroxysms are often excited by eating or drinking anything cold, by a draught of air, or by imitation; they are usually more frequent during the night than the day, and in a close room than in the open air.

In less severe cases no paroxysms of the grade above described may occur, and no typical whoop may be heard throughout the attack; but the paroxysmal nature of the cough which continues until the plug of mucus is expelled, the watery eyes, and the vomiting which follows a paroxysm, stamp the disease as pertussis. In young infants the whoop is frequently not marked. The child sometimes coughs until he is asphyxiated, and yet no whoop occurs. The paroxysms are also modified by intercurrent disease, especially by attacks of pneumonia or severe bronchitis. At such times they usually become less frequent and less typical, and may be absent for several days, returning as the complication subsides.

The seat of the irritation which produces the cough has been variously located by different observers. Some have thought it to be in the nose, others in the trachea, the bronchi, or the larynx. It is very prob-

able that it may not always be in the same place and that the infectious catarrh, which is really the most important element in the disease, may vary in its intensity and location in different cases. The weight of evidence seems to be that in the great majority of cases the source of irritation is in the larynx or trachea. From laryngoscopic examinations made during the disease, Von Herff found the mucous membrane of the larynx to be swollen and congested, and occasionally the seat of small hemorrhages or superficial ulcers. He states that the frequency and severity of the paroxysms corresponded with the degree of laryngitis, and he found that a paroxysm could always be excited by irritating the mucous membrane between the arytenoid cartilages. During a paroxysm he observed that there was a collection of mucus on the posterior laryngeal wall, the removal of which had the effect of shortening the paroxysm.

Rosslach made laryngoscopic examinations, with negative results so far as the larynx was concerned, but he states that a plug of mucus could always be seen in the lower trachea for one or two minutes before the paroxysm occurred. There is little doubt that this collection of mucus is the exciting cause of the paroxysm, as it is a familiar clinical fact that the paroxysm continues until this is dislodged.

The average duration of the spasmodic stage is about one month. It increases in intensity for the first two weeks, remains stationary for about a week, and then gradually diminishes in severity. The course and duration of this stage are, however, subject to wide variations. In mild cases it may last only a week; in severe cases, especially in the winter season, it may continue for three months, at times almost subsiding, but lighting up again with all its previous severity with every fresh attack of cold. After it has entirely ceased the whoop may return with an attack of bronchitis, and continue for a month or more. This is not to be regarded as a true relapse of pertussis. The habit of the paroxysmal cough once established, it tends to recur with every slight bronchitis, often for months afterward.

*The Stage of Decline.*—Gradually the severity of the paroxysms abates, the whoop ceases, and the cough resembles more and more that of ordinary bronchitis. This stage usually continues about three weeks, but may be prolonged indefinitely in the winter months.

**Complications.**—*Hæmorrhages.*—The hæmorrhages of pertussis are mechanical, and depend upon the intense venous congestion which accompanies the paroxysm. Epistaxis is the most frequent variety, and occurs in a considerable proportion of the severe cases, in a few with almost every severe paroxysm, but it is rarely severe enough to require local treatment. Hæmorrhages from the mouth may have their origin either in the pharynx or the bronchi, the blood being brought up by the cough; such hæmorrhages are usually small. Conjunctival hæmorrhages are less frequent, and are usually slight, although I have seen the entire



conjunctiva covered. In a case under my observation there was bleeding from both ears with every severe paroxysm, for more than a week. This child had previously suffered from scarlatinal otitis, with perforation of the drum membrane. Small extravasations into the cellular tissue beneath the eyes are occasionally seen, giving an appearance somewhat like an ordinary "black eye." Intracranial hæmorrhages are not frequent, but many examples have been recorded, and they may be severe enough to produce death. They are usually meningeal, very rarely cerebral; according to their extent and location they may produce hemiplegia, monoplegia, aphasia, facial paralysis, or disturbances of sight, hearing, or sensation; in addition, there may be convulsions or rigidity, but rarely complete coma. The extravasations are sometimes small and the symptoms which they produce may disappear at the end of a few weeks. More extensive hæmorrhages may cause death. In almost every instance these hæmorrhages have occurred as a direct result of the severe paroxysms. Purpura hæmorrhagica is occasionally seen as a sequel of pertussis.

*Respiratory System.*—The most serious complications of pertussis are connected with the lungs. By far the largest proportion of deaths is due to pulmonary complications, usually broncho-pneumonia. This is more frequent in winter and spring than in the summer months, and is especially to be dreaded during infancy. In later childhood lobar pneumonia is occasionally seen. Pneumonia rarely begins before the second week of the disease, and most frequently develops at the height or toward the close of the spasmodic stage. The physical signs present no peculiarities; the cough changes somewhat in character during the pneumonia, and the whoop may not be heard. The prognosis of the pneumonia is bad, because of the debilitated condition of the children at the time of its occurrence. A great danger is from the supervention of convulsions, this being a frequent mode of termination. As there is always considerable emphysema the rapidity of breathing is frequently out of proportion to the temperature, which often is only moderately elevated. If the child escapes the dangers of the acute stage, death may still occur from exhaustion, owing to the protracted course which the disease frequently runs.

Bronchitis of the large tubes is present in almost all the severe cases, and is not of itself serious. Bronchitis of the small tubes has the same dangers and the same complications as broncho-pneumonia.

Vesicular emphysema has been present, I think, in every case which I have seen upon the post-mortem table; a certain amount of it, no doubt, occurs in every severe case. It is produced by the forcible cough of the paroxysm. In very severe cases interstitial emphysema is also found. Rupture of the air-blebs which form on the surface of the lung may lead to emphysema of the cellular tissue of the mediastinum, and

pertussis are hernia, prolapsus ani, and ulcer of the frenum

**Prognosis.**—The only constant features of pertussis are the course of the disease and its communicability. In many cases the typical whoop is heard. There are no symptoms by which a positive diagnosis is made in the catarrhal stage; but a cough not accompanied by fever or physical signs, which steadily increases in severity for two weeks, and which occurs chiefly at night, is always suspicious. When, in addition, the cough begins to come in paroxysms, accompanied by suffusion of the face and occasionally by vomiting, there can be little doubt even though no whoop is heard. If the disease is severe the diagnosis is practically certain. Mild cases which do not even as far as the symptoms mentioned are most puzzling. But if there is a history of exposure, if the cough continues from four to six weeks, little influenced by treatment, and if other cases follow, the disease must be pertussis. Without evidence of communicability, however, the diagnosis may be in doubt even after the disease is over. In early infancy the cough may have more or less of a spasmodic character, and a fairly typical whoop is often heard in the course of an ordinary bronchitis. We have several times seen abortive or very short attacks in one member of a family of children, the others having the disease in a typical form. Occurring by themselves such cases can not be recognised.

Irritation of the pneumogastric or recurrent laryngeal nerve from enlarged tracheal or bronchial lymph nodes, whether of a simple or tuberculous character, may give rise to a spasmodic cough, which in certain cases may be indistinguishable from pertussis. The prolonged duration of these cases is sometimes the only diagnostic point; but the paroxysms are usually not so severe as in true pertussis, and the course is generally less typical.

The presence of a leucocytosis may be of considerable aid in diagnosis.<sup>1</sup>

**Prognosis.**—The most important factor in the prognosis of the disease is the age of the patient. After the fourth year it is indeed rare that either a fatal result or serious complications are seen; but during infancy, and particularly during the first year, there are few diseases more to be dreaded. This is especially true on account of the connection of whooping-cough with the three most fatal conditions of infancy

<sup>1</sup> Fröhlich and Meunier first called attention to the leucocytosis accompanying pertussis, far exceeding that of any other afebrile disease of the respiratory tract. It appears in the early part of the convulsive stage, and disappears slowly with improvement. The count is usually between 15,000 and 25,000, although it may reach 50,000. There is an increase in the lymphocytes at the expense of the neutrophils. The lymphocytes may form 60 to 80 per cent of the total leucocytes. The leucocytosis is little influenced by complications, and even during broncho-pneumonia the lymphocytes continue to be in excess.

—broncho-pneumonia, diarrhœal diseases, and convulsions. Fully two-thirds of the deaths from whooping-cough occur during the first year of life. The prognosis is very much worse in infants under three months than in those who are older and consequently have more resistance. It is better in the summer than in the winter, because broncho-pneumonia is then less frequent. It is particularly bad in delicate infants, in those who are rachitic, in those who are prone to attacks of bronchitis, in those who have suffered previously from pneumonia, and in those with a strong tendency to tuberculosis.

The exact mortality of whooping-cough it is difficult to state in figures. During the first year of life it is probably not far from twenty-five per cent, although it diminishes rapidly after this time. In foundling asylums and hospitals for infants it is to be ranked among the most fatal diseases, and in some epidemics the mortality in such institutions is as high as fifty per cent.

Fully two-thirds of the deaths during whooping-cough are from broncho-pneumonia; the next most frequent cause is diarrhœal diseases. Convulsions may be the mode of death in either of the above conditions, or may occur apart from them. During the first year, death often results from marasmus, the child having been reduced by the prolonged disease. Occasionally death is due to asphyxia following a severe paroxysm, to intracranial hæmorrhage, or to general emphysema.

As a predisposing cause of tuberculosis, pertussis is second only to measles. In both diseases tuberculosis develops in much the same way and from practically the same causes.

**Prophylaxis.**—Pertussis is a contagious disease, and a child suffering from it should be isolated from other children whenever this is possible. Children with pertussis should never be allowed to attend school, and needless exposure should always be avoided.

Young infants, delicate children, and those with a predisposition to tuberculosis, should be most carefully protected against exposure, since it is in them chiefly that the disease is likely to be serious. As it is from the patient that the disease is nearly always contracted, there does not exist the same necessity for the fumigation and disinfection of apartments as after other contagious diseases. In institutions, however, this should always be practised, and in private houses if the room is subsequently to be occupied by an infant.

It is as undesirable as it is impossible to confine a child with pertussis to a single room during the attack; all those persons for whom exposure would be dangerous should therefore be sent away from the house. Quarantine should continue for at least six weeks, or until the spasmodic stage is over.

**Treatment.**—We have as yet no specific remedy for pertussis. The important thing in most cases is the hygiene or general management of

the case; fully half of the cases seen in practice require nothing more. Much harm is done by indiscriminate drug giving.

*General Measures.*—Fresh air is important throughout the attack. It is almost invariable that the paroxysms are fewer while patients are out of doors, and more frequent when they are in close rooms. Older children with pertussis may go out even in winter except on stormy, raw, or windy days. With infants and delicate children, the outdoor treatment in cold weather so enthusiastically advocated by some writers should be used with the greatest caution. It should certainly not be permitted if the patient has even the slightest amount of bronchitis. My own experience is that during the winter in a climate like that of New York or New England, the class of patients just referred to are better off indoors, taking their airing, if at all, in their rooms. In warm weather or in a mild climate all children should be kept in the open air as much as possible.

A change of climate is desirable when the cough is unduly prolonged, also for delicate children in winter. A warm place at the seashore is one which is most likely to be beneficial. The improvement following a sea voyage is often very marked, surpassing even a residence at the seashore.

The rooms occupied by children suffering from pertussis should be frequently changed, thoroughly aired, and occasionally fumigated. A change of rooms, clothing, bedding, etc., sometimes exerts a marked influence on the course of very prolonged attacks, the inference being that continued re-infection takes place. Such a change should be made twice a week, and it is of special importance in hospitals, where many children quarantined in a ward seem to cough interminably.

Careful feeding and attention to the bowels are matters of the greatest importance; with infants particularly, chronic indigestion and abdominal distention have a very marked effect in increasing the frequency of the paroxysms. The abdominal support furnished by a snugly fitting band, adds materially to the comfort of the patient in a severe attack. Feeding is difficult since vomiting occurs so easily. In most cases it is necessary to repeat the meal in a short time, if the first one has been vomited. Children over two years old should in all such cases be kept upon a fluid diet, chiefly of milk. For infants, milk should be diluted, and in many instances it should also be partially peptonised. Any medication which causes disturbance of the stomach should be omitted.

*Local applications* to the rhino-pharynx or to the larynx may be made by means of a spray or swab. Resorcin and carbolic acid, each in a one-per-cent solution, are most used. These applications are made once or twice daily. I have never seen from any of the above methods the beneficial results claimed, and I believe them to have been exaggerated. The

application of cocaine to the larynx should never be employed in young children on account of the danger of poisoning.

Inhalations are of much more value. They are useful to modify the catarrh by allaying irritation, facilitating the expulsion of the mucus, and possibly as antiseptics. Those most employed are carbolic acid, creosote, and cresolene. In my experience creosote is the best. These substances may be used upon cotton in a respirator, or vapourised over an alcohol lamp. The possibility of absorption should not be forgotten, and the urine should be watched. Where the paroxysms are frequent and of great severity, chloroform may be used to ward off convulsions or prevent dangerous asphyxia. In such conditions O'Dwyer used intubation with striking benefit. The tube entirely overcomes the glottic spasm which is the chief cause of suffering and danger.

*Internal Medication.*—Of the innumerable drugs which have been recommended for this disease, there are two which possess undoubted advantages over all others, viz., belladonna and antipyrine. In giving belladonna it is important to begin with a small dose and gradually increase both its frequency and size until the physiological effects of the drug are produced. To an infant two years old, one-fourth of a minim of the fluid extract may be given every four hours as an initial dose, gradually increasing to every two hours; if atropine is used, gr.  $\frac{1}{16}$  may be given in the same way. Although belladonna usually has a decided influence in reducing both the frequency and the severity of the paroxysms, it causes many unpleasant symptoms, and its effects must be closely watched.

Antipyrine has been in my experience more generally useful than any other single drug. It may be given with safety, even to young infants, in considerably larger doses than are ordinarily employed. For a child six months old the initial dose may be one grain every three hours; later this may be given every two hours. For a child two years old the initial dose may be two grains repeated every four to six hours, gradually increasing up to two grains every two hours. Should pneumonia develop, the antipyrine should be discontinued. A combination of the bromide of sodium with antipyrine is often better than the latter given alone.

Nearly all drugs which allay nervous irritability have a certain amount of effect in controlling the paroxysms of pertussis; codeine, chloral, and trional are useful where the night attacks are so severe as to prevent sleep. I do not believe that any form of internal medication or local treatment shortens pertussis; but, inasmuch as the disease is self-limited, great benefit to the patient results from the reduction of the number and the diminution of the severity of the paroxysms.

In establishing the value of any method of treatment, it should be remembered that the number of cases in which the duration of the disease

is short is large, and also that almost any method of treatment if employed after the attack has reached its height will be thought beneficial, as the natural tendency is then to improve. The value of any particular line of treatment is to be judged in a given case only by its effect in reducing the number and severity of the paroxysms. This ought to be evident in the case of drugs within two or three days, and can only be determined by keeping a careful record of the number of severe paroxysms day and night. No drug succeeds equally well in all cases.

In a mild case, where the number of paroxysms does not exceed eight or ten during the day, where there is no vomiting and the general health is not affected, it is not usually advisable to continue the administration of any drugs throughout the disease. A single dose of antipyrine or codeine at night may be all that is necessary. All cases in infants must be watched with great care and the parents warned of the possible dangers which may supervene suddenly, even in the course of mild attacks. For severe cases antipyrine should be given to diminish the frequency and the severity of the paroxysms, and inhalations of creosote used if much catarrh is present. All the fresh air possible should be allowed. For older children the same plan of treatment may be followed, or quinine or belladonna may be substituted for the antipyrine.

As these drugs are given solely for the purpose of diminishing the frequency and severity of the paroxysms, their continuous use should be deferred until the symptoms are sufficiently severe to greatly disturb the child, the benefit at this period being more striking than if they are begun early and used continuously.

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## CHAPTER VII.

### MUMPS.

#### (*Epidemic Parotitis.*)

MUMPS is a contagious disease characterised by swelling of the parotid, and sometimes of the other salivary glands, with constitutional symptoms which are usually mild. Both severe complications and a fatal termination are extremely infrequent. The disease is not a very common one, and general epidemics are rare.

**Pathology and Lesions.**—The contagious character, definite incubation, and typical course, stamp the disease as a general one due to a specific organism. This is probably a very minute Gram-negative diplococcus. It can be demonstrated in Steno's duct, in the testicles when epididymitis is present, and frequently in the blood. It is probable that infection takes place through the salivary ducts.

The precise nature of the changes in the gland is still a matter of

dispute, as opportunities for pathological examination are very rare. From existing evidence it would appear that the gland substance is first involved, and afterward the surrounding connective tissue. The gland is the seat of an intense hyperæmia and œdema; the walls of the salivary ducts are swollen, and the ducts are obstructed. While the primary disease does not tend to excite suppuration, pyogenic germs may occasionally gain entrance and an abscess form; but this is to be regarded as a rare accidental infection.

In the great proportion of cases the parotids alone are affected, although the same changes are occasionally found in the other salivary glands. There are no other essential lesions of the disease, those which are found depending upon complications.

**Etiology.**—Mumps is spread by contagion, close contact being usually required to communicate the disease, although it is known to have been carried by a third person and even by clothing. The susceptibility of children to the poison of mumps is much less than is the case with the other contagious diseases, so that only a small number of those who are exposed take the disease. The greatest predisposition is between the fourth and fourteenth years. Infants are rarely affected, although a case in a child three weeks old is vouched for by so good an observer as Demme.

Mumps is contagious from the beginning of the symptoms. Two cases have come under my notice in which the disease was communicated before any swelling was seen. It is impossible to fix with certainty the duration of the infective period. The disease is undoubtedly communicable for several days after the swelling has subsided; and for safety a case should be isolated for three weeks from the beginning of symptoms, or at least ten days after the swelling has disappeared.

**Incubation.**—In forty-eight collected cases in which the incubation was definitely determined, it varied between three and twenty-five days. It was less than fourteen days in only four cases, and in twenty-six of the forty-eight cases it was between seventeen and twenty days. In three cases of my own in which it could be definitely fixed, the incubation was nineteen days in one case and twenty days in two cases. The average period of incubation, then, may be stated to be from seventeen to twenty days.

**Symptoms.**—In the milder cases the local symptoms are the first to attract attention; in those which are more severe there are frequently prodromal symptoms of from twelve to forty-eight hours' duration—anorexia, headache, vomiting, pains in the back and limbs, and fever. Soltmann has reported a case ushered in by convulsions. The initial temperature in a mild attack is 100° to 101° F.; in a severe one, from 102° to 104° F.

Of the local symptoms, the pain usually precedes the swelling; it is

increased by movement of the jaws, by pressure, and sometimes by the presence of acid substances in the mouth. It is usually referred to the posterior part of the jaw just below the ear. The swelling may begin simultaneously in both parotids, but more frequently one side is involved a day or two in advance of the other. It usually reaches its maximum on the third day, often on the second, remains stationary for two or three days, and then subsides gradually. The degree of swelling varies with the severity of the attack. When it is marked, the patient may be so changed in appearance as scarcely to be recognisable; it fills the lateral region of the neck between the jaw and the sterno-mastoid muscle and extends forward upon the face to the zygomatic arch, so that the centre of the tumour is usually the lobe of the ear. The other salivary glands may swell simultaneously with the parotids, or several days later, even after the parotid tumour has disappeared. Occasionally swelling of the submaxillary or the sublingual glands occurs before that of the parotid, and in rare instances these may be the only glands affected.

As a rule, the parotid of both sides is involved. Of 282 cases both sides were affected in 215. When one side alone is involved, it is the left a little more frequently than the right. The interval between the swelling of the two sides may be a week, or even five or six weeks, but usually it is only two or three days.

The salivary secretion is usually very much diminished, and the dry mouth causes great discomfort. An exceptional instance has been reported by Simon, in which a distressing salivation occurred, the secretion amounting to six or eight ounces daily.

Although as a rule the patient is not seriously ill, mumps may in rare cases produce most alarming and even dangerous symptoms. The temperature may for several days reach  $104^{\circ}$  F. or more, deglutition may be extremely difficult, pressure on the jugular veins may lead to venous hyperæmia of the brain, causing headache and sometimes delirium; there is sometimes great prostration and the symptoms of the typhoid condition. These severe attacks are nearly always in children over twelve years old.

The constitutional symptoms of mumps usually last from three to five days; the swelling continues on an average a little less than a week. If the case has been a severe one, slight swelling may continue for two weeks or even longer. Relapses, in which the opposite side from the one first affected is involved, are quite frequent, occurring in about ten per cent of the cases.

**Complications and Sequelæ.**—In childhood the complications are few and usually unimportant; but in adolescence they are occasionally serious. Orchitis is exceedingly rare in childhood; of 230 cases observed by Rilliet and Barthez, this was seen in but ten, and only three of these cases were under fifteen years, and no case under twelve years old. When



orchitis occurs it is generally toward the end of the second or the beginning of the third week; it is usually marked by an accession of fever, sometimes by a chill; if severe, nervous symptoms may be present. The body of the testicle and not the epididymis is generally affected. The acute symptoms continue for three or four days, and the entire duration of the attack is about a week; although the testicle is often enlarged for some time afterward, and atrophy of the organ may follow.

In females, congestion and swelling of the breasts, ovaries, or labia majora may occur; and, although these complications are all very rare, most of them have been observed even in young children.

Nephritis has in a few instances followed mumps, sometimes coming on as late as four or five weeks after the attack. Single cases have been reported by Croner, Isham, Hensch, and others. Nervous sequelæ are more frequent, but even these are rare. I have seen a case of multiple neuritis in a boy of twelve which developed two weeks after a severe attack of mumps. The paralysis was general, lasted for six weeks, and was followed by complete recovery. Jaffrey has reported a similar case. Facial paralysis three weeks after mumps has been reported by Hillier, apparently due to an extension of inflammation from the gland to the seventh nerve.

Pearce has collected an interesting series of forty cases of deafness following mumps, in which there was no sign of otitis, the symptoms coming on suddenly with vertigo, a staggering gait, and often with vomiting. In most of the cases the deafness was unilateral and the loss of hearing was permanent. The cause assigned was disease of the auditory nerve, the seat of the trouble being in the labyrinth. Toynbee has reported an instance of hæmorrhage into the labyrinth. Otitis media is rarely seen.

Suppuration of the parotid gland occurs in about one per cent of the cases, and is probably due to accidental infection. Gangrene and sloughing of the parotid were observed twice by Demme in 117 cases; both of these proved fatal. Pneumonia, meningitis, endocarditis, and pericarditis have been observed as complications of mumps, although all are extremely rare.

**Prognosis.**—In the great proportion of cases mumps is a mild disease, and terminates in complete recovery in a few days. In young children complications are infrequent, and those which occur are rarely severe.

**Diagnosis.**—Mumps is most likely to be confounded with acute swelling of the cervical lymph nodes. In a parotid swelling, the lobe of the ear is near the centre of the tumour, which extends backward to the sterno-mastoid muscle and forward upon the face as far as the zygomatic arch, embracing the angle and ramus of the jaw.

A swollen lymph node is usually entirely below the ear and behind

the jaw, not extending upon the face. The tumour is generally smaller and more circumscribed if only a single node is involved, and it comes on much more slowly than does mumps. When only the submaxillary or sublingual glands are affected, the diagnosis from swollen lymph nodes is sometimes impossible except by the course of the disease. Mumps is characterised by the rapidity with which the swelling occurs, and by its relatively short duration.

**Treatment.**—The disease is self-limited and the individual symptoms rarely distressing, so that in most cases very little treatment is required. If constitutional symptoms are present the patient should be kept in bed, and if there are none he should be confined to the house. The gland should be protected by cotton or spongio-piline, and if the pain is severe heat should be applied. The diet should be liquid, on account of the pain produced by mastication. The mouth should be kept clean by the use of some antiseptic mouth-wash. The general symptoms and complications are to be treated according to the indications presented. Cases of mumps occurring in schools or institutions should be quarantined for three weeks, and in private practice where there are susceptible persons. Fumigation and disinfection after an attack are unnecessary.

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## CHAPTER VIII.

### DIPHTHERIA.

DIPHTHERIA may be defined as an acute, specific, communicable disease due to the bacillus of Klebs and Loeffler. It is usually characterised by the formation of a false membrane upon certain mucous membranes, especially those of the tonsils, pharynx, nose, or larynx. Like other pathogenic organisms, however, this germ acts with varying intensity, and may cause inflammation of all degrees of severity, from a mild catarrhal angina to the most serious membranous inflammation; but to all alike the term diphtheria should be applied. In its mild form it may be almost without constitutional symptoms; but in its severe form it is attended by great general prostration, cardiac depression, and anæmia, it is frequently complicated by pneumonia and nephritis, and it may be followed by localised or general paralysis; it then constitutes one of the diseases most to be dreaded in childhood.

**Etiology.**—*The Bacillus Diphtheriæ.*—This was first described by Klebs in 1883, and during the following year it was isolated by Loeffler and shown to be pathogenic. It varies considerably in size and shape even in the same culture. In a specimen it occurs singly or in pairs, sometimes in chains of three or four; the bacilli may lie parallel, but frequently two form an acute or an obtuse angle. They are straight or

slightly curved, and sometimes branching; they may be swollen or club-shaped at their ends.

*Distribution and Mode of Communication.*—In most large cities diphtheria prevails endemically, with periods in which outbreaks of considerable severity are observed. In the country it prevails chiefly as an epidemic. The disease is often introduced into remote districts in some inexplicable manner, and before its nature is recognised a large number of persons may be exposed, and an epidemic results.

Diphtheria does not arise *de novo*. Every case has its origin in a previous case either directly or remotely. The bacilli may enter the body through the inspired air; they may be taken into the mouth with toys or other articles upon which they have lodged, or by kissing, and sometimes by accidental inoculation. As a rule, the bacilli first gain a foothold upon the mucous membrane of the tonsils, nose, or larynx.

Direct infection is the cause in the great majority of the cases. There is no proof that the bacilli are contained in the breath of a person suffering from the disease. They are present in great numbers in the saliva and mucus from the mouth and nose, often being distributed by sneezing, coughing, or even by talking. They are contained in pieces of membrane which are discharged; they are not present in the urine or fæces. The most contagious cases are those of pharyngeal diphtheria on account of the amount of discharge which accompanies them. The least contagious are those in which the membrane is limited to the larynx and lower air passages.

Direct infection may occur from persons convalescent from diphtheria, whose throats still contain virulent bacilli, or from persons suffering from a mild form of the disease, which is not recognised as diphtheria. In the latter way it is often spread in schools. It has been repeatedly shown that a person may harbour virulent bacilli in his nose or throat, and may even communicate the disease to others, without himself suffering from diphtheria at any time.

The length of time during which a patient with diphtheria may convey the disease to others is somewhat uncertain. Transmission is possible so long as virulent bacilli remain in the throat; these are frequently found two weeks after the membrane has disappeared and the patient is regarded as entirely well, and in a few cases they are found five or six weeks or longer after recovery.

Indirect infection is not uncommon, and may occur from the bed or clothing of the patient, from the carpet, furniture, wall-paper or hangings of the room, from toys or picture-books, from dishes, feeding bottles, or drinking-cups, from swabs and brushes used for local applications to the throat, from spoons and tongue-depressors, and from surgical instruments with which tracheotomy or intubation has been done. Diphtheria may be carried by a third person, but rarely except by one who

has been in close contact with the patient—either the physician or nurse. The frequency of diphtheria in physicians' families bears witness to the great danger of infection in this manner.

Bacilli may retain their virulence for an indefinite period. Both Park and Loeffler found cultures in blood-serum to be virulent after seven months; Roux and Yersin, bacilli in dried membrane to be virulent after twenty weeks; and Abel, upon a child's toy after five months.

Domestic animals may in rare instances be carriers of infection, and in the case of pigeons, at least, they may themselves suffer from the disease. Diphtheria has been repeatedly spread by milk, but very rarely through the contamination of a water supply.

*Predisposing Causes.*—Local conditions in the throat influence very largely the occurrence of diphtheria. An important predisposing cause is the existence of a chronic catarrhal inflammation of the mucous membranes of the nose and throat, so frequently found in children suffering from adenoid growths of the pharynx or from enlarged tonsils. These adenoid growths, the tonsillar crypts, and the cavities of carious teeth, may harbour the bacilli for a considerable time both before and after an attack. The condition of the mucous membranes of the nose and pharynx in other acute infectious diseases furnishes a marked predisposition to diphtheria. This is most striking in the case of measles and scarlet fever; it is seen less frequently in typhoid fever and influenza.

The two sexes are about equally liable to the disease. Children under ten are much more often affected than those who are older, the greatest susceptibility as regards age being between the second and fifth years.

While diphtheria is seen throughout the year, it is more frequent during the cold than the warm months.

The incubation of diphtheria is short. In most of the cases in which it could be definitely traced it has been between two and five days. The virulence of the bacillus varies much in different cases and in different seasons, and while it is frequently true that persons infected from a mild type of the disease have a mild attack, and those infected from a malignant one a severe attack, there is no certainty that such will be the sequence. Park states that, out of many hundreds tested in the laboratory of the New York Health Department, by far the most virulent bacillus was obtained from the throat of a boy who had what was clinically a very mild form of tonsillar diphtheria.

The immunity conferred by one attack of diphtheria is not of long duration, amounting probably to a few months only; but the passive immunity conferred by antitoxine is still shorter, lasting but a few weeks. In patients therefore to whom antitoxine has been given, a second attack may occur after a very brief time.

**Lesions.**—The essential lesions of diphtheria consist not in the production of a membrane, but, as long ago pointed out by Oertel, in certain acute degenerative changes in the cells of the body caused by the diphtheria toxins. These changes are seen particularly in the epithelial cells of the affected mucous membranes, the heart muscle, the kidney, the liver, the central and peripheral nervous system, the spleen, and the lymph glands. There are other lesions which are the result of the action of other organisms, especially the streptococcus pyogenes and the pneumococcus, either alone, together, or in conjunction with the diphtheria bacillus. The most important lesions due to these organisms are bronchopneumonia and nephritis; but there may be found in the blood, and in many of the organs of the body, the evidences of the invasion of these bacteria, i. e., a streptococcus septicæmia, less frequently a general pneumococcus infection.

**Distribution of the Diphtheria Bacillus in the Body.**—Unlike many other pathogenic organisms, the diphtheria bacillus is not in most cases widely distributed throughout the body. It is found in great numbers on the surface of the affected mucous membranes and in the false membrane itself, particularly in its superficial portion, but it does not invade deeply the subjacent structures.

The frequency with which the diphtheria bacillus and other organisms are found in the blood and viscera is shown in a series of 209 autopsies studied by Councilman, Mallory, and Pearce, of Boston, in 1901. The following table shows the percentage of cases in which the different bacteria were found by culture:

|                            | Heart's blood. | Liver.       | Spleen.      | Kidneys.     |
|----------------------------|----------------|--------------|--------------|--------------|
| Diphtheria bacillus.....   | 6 per cent.    | 20 per cent. | 12 per cent. | 19 per cent. |
| Streptococcus.....         | 20 “           | 30 “         | 27 “         | 28 “         |
| Staphylococcus aureus..... | 2.5 “          | 4 “          | 3 “          | 8 “          |
| Pneumococcus.....          | 1.5 “          | 2.5 “        | 1.5 “        | 5 “          |

In this series, 153 cases were pure diphtheria; 56 were complicated by measles or scarlet fever or both. The streptococcus was much oftener found in the viscera in the complicated cases; otherwise there was little difference in the two groups of cases.

**The Diphtheria Toxines.**—The wide-spread effects seen in diphtheria are due to the action of certain substances called *toxines* which the diphtheria bacillus produces during its growth on mucous membranes. They are very diffusible, readily entering the lymphatic circulation and the blood, and through these channels may affect the entire body. In susceptible animals there may be produced by the injection of these toxins all the characteristic lesions of diphtheria except the membrane, as well as the essential symptoms of the disease, even includ-

PLATE XVIII.

B



A



THE DIPHThERITIC MEMBRANE.

- A. Typical tonsillar diphtheria.  
B. Severe pharyngeal diphtheria (fatal case).  
C. Pseudo-diphtheria. The specimen is seen from behind, the larynx and trachea having been laid open, and shows an extensive membrane involving the epiglottis and the entire lower pharynx, but extending into the larynx only a short distance. It is also seen upon the posterior surface of the uvula and soft palate, the tonsils being only partially covered. The colour of the membrane is not characteristic of pseudo-diphtheria, as the same appearance is often seen in true diphtheria, particularly of the septic type.



ing paralysis. For the production of the membrane living bacilli are required.

*Catarrhal Diphtheria.*—The routine practice of making cultures from diseased throats has established the fact that catarrhal inflammation may often be the only result of diphtheritic infection. Although to the naked eye there were only the ordinary changes of a simple inflammation, Oertel found the characteristic degenerative changes in the epithelial cells, varying in degree with the severity of the process.

*The Diphtheritic Membrane.*—The membrane in diphtheria is most frequently seen upon the mucous membrane of the tonsils, soft palate, uvula, pharynx, nose, larynx, trachea, and bronchi; less frequently upon the mouth, lips, œsophagus, conjunctivæ, middle ear, stomach, and genital organs. It may also affect fresh wounds, notably a tracheotomy wound, or any abraded cutaneous surface. The gross appearance of the membrane varies greatly (Plate XVIII). It is most frequently of a gray or mouse-colour, but it may be pearly white, yellow, green, and sometimes almost black. It is composed of fibrin, cells, granular matter, and bacteria. Its consistency varies with the relative proportions of the different elements. When made up chiefly of fibrin it is firm and retains its form, often being discharged as a complete cast of the nose, larynx, or trachea. When the amount of fibrin is small the membrane is soft, friable, and sometimes granular. It is more closely adherent upon the mucous membranes covered with squamous epithelium, as in the pharynx and upper air passages, than upon those covered with columnar and ciliated epithelium, as in the lower air passages.

The microscopical examination shows the fibrin to be sometimes granular, but usually in the form of a network, inclosing in its meshes small round cells and epithelial cells in various stages of degeneration. On the surface and in the superficial layer there is usually found quite a variety of bacteria including diphtheria bacilli. Beneath this is a cellular layer containing little or no fibrin, in which also the diphtheria bacilli are usually found. In the deepest parts of the false membrane and in the mucous membrane itself the bacilli are few in number or absent.

Changes which are similar in all the affected mucous membranes, are found in the epithelial cells which undergo marked degeneration with fragmentation of their nuclei; the mucosa is infiltrated with leucocytes. The infiltration with small round cells is variable in degree in the different mucous membranes; in some it extends deeply into the submucous and even the muscular layers, while in others it is very superficial. Marked evidences of degeneration are seen also in the cells infiltrating the deeper layers. In places the epithelium is detached, in others the line between the false membrane and the granular mucous membrane is scarcely distinguishable.



*The Seat and the Distribution of the Membrane.*—This varies somewhat with the age of the patient, the season, and the peculiarity of the epidemic.

My own records show that the larynx is involved in about forty per cent of the cases in children under three years. In general the statement may be made that the younger the child the greater the liability of the disease to attack the larynx; also when the larynx is affected, the greater the tendency to spread to the trachea and bronchi. The larynx and lower air passages are rather more frequently attacked in winter than in summer.

The tonsils are the most frequent and usually the earliest seat of the diphtheritic membrane; it may form here a tough, leathery patch, partially or completely covering and very adherent to them; or the disease may affect only the tonsillar crypts, so that the gross lesion may resemble that of ordinary follicular tonsillitis. There is in most cases only moderate swelling, but it may be so great that the tonsils are in contact. The surrounding cellular tissue is infiltrated with inflammatory products.

The membrane covering the pharynx and uvula is also usually very adherent and intimately blended with the mucous membrane. The uvula is swollen and œdematous. Membrane may be seen only upon the fauces and uvula, or the posterior and lateral pharyngeal walls may be covered down to the level of the cricoid cartilage, but generally not below this point. If the posterior pharyngeal wall is covered, the membrane is apt to extend into the rhino-pharynx, and may fill the entire pharyngeal vault, covering the posterior portion of the velum and extending into the posterior nares. The adenoid tissue of the vault is frequently the part most affected.

The nose may be involved secondarily to the rhino-pharynx, or the infection may be through the anterior nares; if the latter, it is not infrequently the only part involved. Many cases classed as nasal are really rhino-pharyngeal. The membrane in the pure nasal cases is usually thick and tough and often separates *en masse*. Both sides are generally involved, but it may be unilateral.

The observations of Councilman, Mallory, and Pearce have shown that it is very common for the accessory sinuses of the nose, especially the antrum of Highmore, to be involved in fatal cases. It seems highly probable that infection of these parts explains the remarkable persistence of diphtheria bacilli in the nose which is occasionally seen.

The epiglottis is swollen to three or four times its normal thickness, and the aryteno-epiglottic folds are œdematous. The anterior surface of the epiglottis is rarely covered by membrane; but its lateral borders and posterior surface, and the aryteno-epiglottic folds are involved in most of the severe pharyngeal cases (Plate XVIII, C). This lesion is associated with pharyngeal rather than with laryngeal diphtheria.

The lesions which extend most deeply are thus seen in the tonsils, uvula, pharynx, and epiglottis. But even here there is very rarely deep or extensive sloughing.

The lesions of the larynx, trachea, and bronchi are similar to the above, although much more superficial. The interior of the larynx may be completely covered, the membrane coating the true and false vocal cords and lining the ventricles of the larynx. The membrane in the larynx is not usually very adherent, and it frequently separates and is coughed up in large pieces or even as a cast. That covering the epiglottis and the aryteno-epiglottic folds is very adherent, like that in the pharynx. Catarrhal laryngitis is not an uncommon complication of pharyngeal diphtheria.

In a considerable number of cases the membrane stops abruptly at the lower border of the larynx. In the trachea it is generally loosely attached, and often it is found at autopsy entirely separated from the mucous membrane. It is almost invariably associated with membrane in the larynx. Usually the membrane in the bronchi is continuous with that in the trachea. Occasionally I have seen the trachea and larger bronchi passed over and found membrane only in the larynx and smaller bronchi. As a rule, the bronchi of both sides are affected, and to the same degree. I once saw a case of laryngeal diphtheria in which membrane was found only in the bronchi of one lung. The above exceptions are to be explained as accidents in the mechanical transportation of bacilli.

The extent of the membrane varies greatly in different cases. It may stop at the bifurcation of the trachea or at the bifurcation of the primary bronchi; but if it goes beyond this point it is likely to extend to the minutest subdivisions. Exceptionally a very tough fibrinous membrane forms in the trachea and bronchi, of sufficient thickness and consistency to be expelled as a cast, reproducing almost the entire bronchial tree.

The inflammation of the mucous membrane of the larynx, trachea, and bronchi is very much less severe and more superficial in character than that of the pharynx, tonsils, and upper air passages.

The buccal cavity is very seldom covered by the membrane; but in the worst cases of pharyngeal disease it may line the cheeks, cover the lips, gums, and more or less of the hard palate, but rarely the tongue. It usually occurs in patches rather than as a continuous membrane. In one case I saw the membrane on the lower lip, extending on to the face, though the buccal cavity was free. It is not common for the diphtheritic membrane to spread down the digestive tract. In 127 autopsies studied by Councilman, Mallory, and Pearce, in which the extent of the membrane was carefully noted, it was found twelve times in the œsophagus, five times in the stomach, and once in the duodenum. The

amount of membrane varied from small striations on the folds of the stomach or œsophagus to a complete covering. The accompanying changes consist in infiltration, hæmorrhage, and cell degeneration. In the intestines there is often found a hyperplasia of the lymphoid elements—solitary follicles and Peyer's patches—with changes similar to those in the lymph nodes elsewhere in the body, but nothing else that is characteristic.

The writers just referred to found otitis, usually double, in sixty per cent of 144 autopsies; although in less than one-third of the number was the complication recognised during life. Mastoid disease is infrequent. Otitis is usually the result of direct extension from the pharynx. It may be due to the diphtheria bacillus alone, to the streptococcus alone, or to both combined; occasionally the staphylococcus or pneumococcus is found. Conjunctival diphtheria is rare and probably due to accidental infection rather than extension through the lachrymal duct. Before the advent of antitoxine, it almost invariably resulted in destruction of the eye; but a number of cases successfully treated have been reported. Diphtheria may attack any muco-cutaneous surface, especially the anus, prepuce, or female genitals; any abraded cutaneous surface, or recent wound, most frequently the tracheotomy wound of the neck. The diphtheria bacilli have been found in pure culture in superficial abscesses.

*Visceral Lesions.*—The visceral lesions<sup>1</sup> of diphtheria are due partly to the action of the diphtheria toxins and partly to the invasion of the body with other organisms, especially the streptococcus. It is to experimental diphtheria that we owe our most accurate knowledge of the former changes, for in human diphtheria the large proportion of all the fatal cases show infection with other organisms, particularly the streptococcus, to a less degree the pneumococcus or staphylococcus. The frequency with which these bacteria are found at autopsy in different organs has been already stated.

The visceral lesions of diphtheria consist in wide-spread areas of cell degeneration similar to those which have already been described as occurring in the epithelial cells of the affected mucous membranes, together with hæmorrhages due to changes in the blood-vessels and possibly in the blood itself.

The lymph nodes of the cervical region are the most constantly and the most seriously affected. Similar but less marked changes are seen in the tracheo-bronchial and the mesenteric groups, and in the lymph nodules of the mucous membrane of the stomach and intestine. There are degenerative changes in the cells of the nodes most affected, with marked infiltration with leucocytes and frequently small hæmorrhages.

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<sup>1</sup> For an exhaustive study of the pathological anatomy of diphtheria, see monograph of Councilman, Mallory, and Pearce (Boston, 1901); being a study of 220 fatal cases.

The cellular tissue in the neighbourhood of the cervical nodes is often extensively infiltrated with cells. The process in the lymph nodes usually terminates in resolution, rarely in suppuration.

The changes in the spleen are quite constant. The organ is swollen, sometimes very much so, and deeply congested. Hæmorrhages are often seen beneath the capsule; the spleen pulp is soft, the follicles are large, and cell degeneration is quite constantly observed similar to that which takes place in the lymph nodes.

There are frequently small hæmorrhages beneath the capsule of the liver, and sometimes these are seen throughout the organ. There are found scattered through the liver, areas of necrotic hepatic cells which are peculiar to this disease; some of these areas are infiltrated with leucocytes.

The kidneys are involved in almost all fatal cases except where death occurs early from laryngeal stenosis, also in nearly every severe case which terminates in recovery. Acute degeneration of the epithelium of the tubes and the tufts is seen in less severe cases and those of shorter duration, and is the direct result of the action of the toxins. In the more severe and protracted cases there is acute diffuse nephritis of variable type and intensity. There is no form of inflammation which is peculiar to diphtheria; in some cases the interstitial changes predominate, in others the glomerular changes. Welch mentions hyaline changes in the glomerular capillaries and small arteries as the characteristic feature of the nephritis of diphtheria.

In children dying suddenly in the early stage of the disease, cardiac thrombi are occasionally found. They may form rapidly only a short time before death, or slowly during several days when the circulation is very feeble. Portions of these thrombi may be carried into the pulmonary or systemic circulation, causing embolism in any of the arteries of the extremities, the lungs, or other viscera. Even in the early fatal cases the heart muscle may be seriously affected; in the later ones this is almost constant. The changes consist in a toxic myocarditis, the left ventricle being most involved. (See Myocarditis.)

Degeneration of the arteries, especially of the endothelial layer, is occasionally seen, and there may be infiltration of the adventitia. The arteries of any of the viscera may be the seat of hyaline degeneration.

Lesions of the brain are rare; both hæmorrhage and embolism may be met with. In the spinal cord and membranes multiple hæmorrhages occasionally occur. The characteristic lesion, however, consists in degenerative changes which are found to some degree in nearly all the more severe cases which have been examined. These affect the ganglion cells of the anterior horns, the anterior and posterior nerve-roots, and sometimes the pyramidal tracts and columns of Goll. Some recent writers are of the opinion that the cord lesions are primary and the degenera-

tion of the spinal nerves secondary. However, the general opinion still prevails that certainly the less severe cases of diphtheritic paralysis are due to peripheral rather than to central lesions. Degenerative changes have been found also in the pneumogastric, spinal accessory, hypoglossal, motor-oculi, and in the cardiac nerves. These nerve degenerations produced by the diphtheria toxine constitute one of the most striking lesions of diphtheria. (See Multiple Neuritis.)

In infants and young children broncho-pneumonia is found at autopsy in fully three-fourths of the cases, and in a large proportion of these it is the cause of death. It is well-nigh constant in cases of diphtheritic bronchitis of the finer tubes, and is usually present where the membrane has extended to the bifurcation of the trachea. The largest factor in the production of pneumonia is the aspiration of diphtheria bacilli and streptococci from the upper air passages; an important part is also played by the pneumococcus and the influenza bacillus. These organisms may be present in many combinations.

With laryngeal stenosis, some emphysema is invariably present, and usually it is of the vesicular variety. In extreme or protracted cases of stenosis there may be interstitial emphysema. Rupture of some of these blebs may lead to the escape of air into the cellular tissue of the mediastinum or of the neck, which may result in the production of a general emphysema of the subcutaneous cellular tissue.

*Blood.*—According to the studies of Ewing, Billings, and others, there is found in all severe cases of diphtheria a reduction in the number of red cells to the extent of 500,000 to 2,000,000. There is a nearly proportionate reduction in the hæmoglobin, this amounting to from twelve to twenty-eight per cent. While the hæmoglobin falls coincidently with the number of red cells, it is regained much more slowly. Leucocytosis is generally present, and usually proportionate to the severity of the attack, but is occasionally wanting in the most severe as well as in some of the very mildest cases. The increase in the leucocytes is in the polymorphonuclear forms. Engel has noted the frequent presence of myelocytes, especially in fatal cases, the proportion of these in some instances reaching sixteen per cent of the white cells. In his observations, every case in which the myelocytes exceeded two per cent, proved fatal.

**Symptoms.**—The clinical picture of diphtheria is one which presents wide variations, depending upon the principal location of the disease, its severity, and its complications. For practical purposes the following seems the simplest grouping that can be made:

1. The mild cases, in which there is either no membrane, or the amount of membrane is small and limited to the tonsils or to the nose, with few or none of the constitutional symptoms which follow absorption of the diphtheria poison. These cases partake essentially of the character of a local disease.

2. The severe cases in which there are marked evidences of constitutional poisoning from diphtheria toxines. This form is usually accompanied by an extensive formation of membrane in the pharynx and sometimes in the nose.

3. The laryngeal cases in which the larynx may be primarily affected or in which it is involved secondarily to the severe pharyngeal form.

4. The malignant cases. In these cases the symptoms of inflammation are especially prominent, not only in the pharynx but sometimes in the lymph glands and cellular tissue of the neck, which may be followed by suppuration or sloughing. This form is frequently complicated by broncho-pneumonia even without laryngeal disease, and sometimes by severe nephritis.

*Cases without Membrane.*—During an epidemic of diphtheria in a family or an institution, cases are frequently seen which present the clinical evidences of only a catarrhal inflammation of the nose or pharynx, and yet cultures show the presence of the diphtheria bacillus. Such cases may be examples of simple catarrhal inflammation with the accidental presence of the diphtheria bacillus; or the inflammation may be caused by infection with the diphtheria bacillus, but not of sufficient intensity to lead to the production of a membrane. The latter is the view of pathologists, and the one to which clinicians must, it seems, inevitably come. However, a membrane has so long been regarded as a *sine qua non* of this disease that the existence of diphtheria without it, is something which the clinician finds it hard to grasp.

Catarrhal diphtheria may be either pharyngeal or nasal. In the pharyngeal cases there are present the usual appearances belonging to a catarrhal inflammation of moderate severity, often accompanied by swelling and tenderness of the cervical lymph glands.

The nasal cases, in my experience, have been most frequent in infants or very young children. Constitutional symptoms may be wanting or so slight as to be overlooked. The only striking thing is a persistent nasal discharge which may be serous and frothy, purulent or bloody. It is usually copious, often excoriating the upper lip and sometimes continuing for three or four weeks before any other symptoms are observed. I have known it to be mistaken for a syphilitic coryza. Such cases can be recognised with certainty only by cultures. Clinical evidence of their true character is sometimes afforded by the appearance of visible membrane in the nose or pharynx, by the development of croup, or by the fact that they cause diphtheria in other children.

Catarrhal diphtheria is not in itself serious, but it may be followed, particularly in young children, by laryngeal diphtheria, or, after it has existed for a time, pharyngeal diphtheria may develop in its usual form. Cases like those just described are to be distinguished from others in

which bacilli, either of the virulent or the non-virulent variety, are found without any evidence of inflammation.

*Cases with a Small Amount of Membrane.—Tonsillar Diphtheria.*—The exudation is usually limited to the tonsils (Plate XVIII, A), and may partake of the character of either follicular or croupous tonsillitis; sometimes there is a slight extension to the faucial pillars or to the pharynx. These cases are quite common, and in some epidemics most of those seen are of this variety. They are more frequent in older children and adults than in infants and young children.

The onset is accompanied by a little soreness of the throat; the initial temperature is from 101° to 104° F.; but the symptoms are often not severe enough to keep the patient in bed. If seen early, the throat shows slight redness, followed by a gray film, and later by a gray or white deposit upon the tonsils. It may start as a small patch which enlarges, or as small, isolated spots which coalesce or remain separate. Until it disappears the membrane generally remains of its original colour. It is generally quite adherent, and can not easily be removed with a swab; usually it is sharply defined, but with a somewhat irregular outline. In many cases the patch is not larger than the finger nail. The inflammatory changes in the pharynx are slight; a faint red arcola is frequently present at the border of the patch. The lymph glands behind the jaw may be slightly swollen. There is no nasal discharge and very little increase in the saliva or mucus from the pharynx. Some constitutional symptoms are present, but they are never severe. The temperature commonly continues above the normal while the membrane lasts, its usual range being from 100° to 102° F. The membrane remains from three to seven days—a shorter time if antitoxine is used. It is very often a matter of surprise that so small an exudate is so persistent. The urine is generally normal. The parents are loath to believe that strict quarantine is necessary in so mild an illness; and when the membrane is only upon the tonsils, even after the disease has run its course, the physician may be led to doubt the diagnosis of diphtheria.

In many cases one with experience can usually make an accurate diagnosis from the clinical symptoms alone; but there are many others in which the diagnosis from ordinary tonsillitis is impossible, even by the most practised observers, except by cultures. When diphtheria bacilli are found in these mild cases the question often arises whether they may not be the non-virulent form. Park tested forty such cases, and found the bacilli to be virulent in thirty-five and non-virulent in five. In twenty of the forty cases the clinical diagnosis was follicular tonsillitis.

*Severe Cases.*—The clinical picture of diphtheria is so modified by the use of antitoxine that those who see it given regularly and early can have but little conception of the horrors of this disease when not thus influenced. The onset in severe cases may be gradual, even insidious.

There is then a slight indisposition for a day or two, and perhaps some soreness of the throat; the temperature may be but little elevated, sometimes less than 100° F. The symptoms may steadily increase in intensity for four or five days, until the maximum is reached. At other times the disease begins abruptly with vomiting, headache, chilly sensations, and a temperature of 103° or 104° F. Occasionally, the first thing to attract attention is the swelling of the cervical lymph glands, which may be so great that mumps is suspected. The abrupt onset is more often seen in young children than in those who are older.

The membrane upon the tonsils resembles that of the mild form previously described, but, instead of remaining limited to them, it gradually spreads to the fauces, the lateral wall of the pharynx, the uvula, the rhino-pharynx, and the posterior nares. The rapidity with which the membrane extends is in direct proportion to the severity of the attack. In some cases it may cover all the parts mentioned in twenty-four hours from its first appearance; in others this may require several days. When the nose is first affected there is an abundant discharge of serum and mucus, occasionally tinged with blood, which may continue some days before any membrane is visible.

When a severe case is fully developed there is a very abundant discharge of mucus from the mouth and nose. The tonsils, the entire faucial ring, and the pharynx are covered with membrane (Plate XVIII, B) which is at first gray and gradually becomes darker, often being of a dirty olive-green colour. Membrane is sometimes seen upon the lips, or in patches in the mouth. There is obstruction to nasal respiration from the swelling of the palate, the tonsils, and the tissues of the rhino-pharynx; the mouth is half open, the breathing noisy, the tongue dry, and the lips are fissured and bleed readily. Occasionally large nasal hæmorrhages occur which may necessitate plugging the nares. Both nostrils are generally blocked by the swelling and the false membrane; the discharge excoriates the upper lip, and frequently has a fetid odour. During the second week there may be regurgitation of fluids through the nose, owing to paralysis of the palate. The lymph glands at the angle of the jaw swell rapidly; in severe cases they are very prominent, and there may also be extensive infiltration of the cellular tissue about them.

The constitutional symptoms usually increase steadily with the extension of the membrane. In the most severe cases the system is overwhelmed with the poison, and all the evidences of intense toxæmia are present by the third day of the disease. This is shown by great muscular weakness and prostration, by a feeble, rapid pulse, and a mental state of complete apathy or stupor, sometimes alternating with great restlessness. It is more frequent for the constitutional symptoms to develop gradually, and not to reach their height before the fourth or fifth day.



The pulse becomes rapid, weak, and compressible, sometimes irregular; and there is a great and steadily increasing anæmia. The course of the temperature is irregular, and bears no constant relation to the severity of the other symptoms. Its usual range is from 101° to 103° F., but in some of the worst cases it may never go above 101° F. It fluctuates irregularly with the development of complications, and sometimes without apparent cause. By the second or third day the urine regularly shows the presence of albumin, and by the end of the first week the quantity is often large. Granular and hyaline casts, and occasionally blood in small quantities, are also found. The amount of urine secreted is not noticeably diminished, and dropsy is rare. There is complete anorexia, and often vomiting and diarrhoea are present; in some of the cases they are prominent. Nervous symptoms are seen in all the very severe cases. There may be dulness and apathy, but more frequently, owing to the discomfort arising from local symptoms, there is extreme restlessness and excitement, sometimes followed by delirium.

At any time during the first week, but not often after that time, symptoms may arise indicating that the disease has extended to the larynx. The first signs of laryngeal invasion usually appear from the second to the fifth day of the disease. These are at first hoarseness, a croupy cough, and slight dyspnoea. In the severe cases these symptoms steadily increase until all the signs of laryngeal stenosis are present.

The local process in the pharynx seems to be a self-limited one, even when no antitoxine is used. It usually reaches its height by the fifth or sixth day, and after that the appearances do not change materially for two or three days. From the seventh to the tenth day, in favourable cases, the diphtheritic membrane begins to loosen and separate from its attachment. It hangs loosely from the palate or uvula, and can often be pulled away in large masses. The detachment is frequently rapid, and in two or three days from the time when the first improvement is seen, the tonsils and pharynx may be almost free from membrane. The mucous surface left behind is of a bright-red colour and bleeds easily. The separation of the membrane in the nose and rhino-pharynx takes place more slowly. From the former it may disintegrate gradually or come away *en masse*. With the disappearance of the membrane the local symptoms abate rapidly—the discharge ceases, the swelling of the lymph glands subsides, deglutition becomes easy and natural, and nasal breathing is re-established. When antitoxine is given the local process passes through similar stages, but much more rapidly.

Simultaneously with these changes in the throat the constitutional symptoms improve, but much more slowly. Convalescence is often protracted. The anæmia and muscular weakness, and most of all the feeble heart action may persist for weeks.

Instead of the usual course just described, the diphtheritic mem-

brane may persist for two or three weeks. In rare cases relapses occur, the membrane forming again after it has entirely or partially disappeared.

The early course of the disease in the fatal cases often does not differ from that of the severe cases which end in recovery, except in the malignant form, which kills in twenty-four or forty-eight hours, and which is rare. In very young children death is most frequently due to broncho-pneumonia, usually accompanying diphtheria of the larynx and bronchi. It may also be due to progressive asthenia the result of diphtheritic toxæmia, or to heart failure, which may come early or late; rarely it is due to nephritis.

*Laryngeal Diphtheria.*—In cases of primary laryngeal diphtheria there are wanting most of the characteristic clinical features which distinguish diphtheria of the pharynx. There are two reasons for this: one is the relatively rapid course of the disease, often producing death from local causes before the constitutional symptoms resulting from the absorption of the toxine have developed; the second reason is, that absorption of the poison by the laryngeal mucous membrane is very feeble as compared with that which takes place from the pharynx. Hence it follows that glandular enlargements, albuminuria and asthenic symptoms are generally wanting; also, that in the cases which come to autopsy early, the parenchymatous degenerations of the heart, kidney, and other organs are seldom found, but instead only such lesions as are connected with the laryngeal disease. The feeble contagion is due to the fact that the course is much shorter, and that the discharge from the nose and mouth is slight, or absent altogether.

In its onset, diphtheria of the larynx is indistinguishable from catarrhal inflammation. It is usually somewhat less abrupt, and apparently not quite so severe for the first twelve hours or even for a longer time. There are present the same hoarse cough and voice, with slight stridor, gradually increasing. The constitutional symptoms are usually not quite so marked, the temperature ranging from 99° to 101° F. The pulse is accelerated, but not weak or intermittent. It is the progress of the disease which indicates its character, usually during the first twenty-four hours. A child beginning in the morning with such symptoms as have been described, may by evening show a decided change for the worse, or the symptoms may increase with great rapidity during the night. At first the voice is hoarse; later it is entirely lost. Dyspnœa in the beginning is scarcely noticeable, but steadily increases hour by hour. Sometimes from the first sign of hoarseness to such extreme dyspnœa as to necessitate intubation may be but a few hours. During the second twenty-four hours all the symptoms are usually well developed. The respiration is often somewhat accelerated, but it may be slower than normal. The face is pale and anxious. The *alæ nasi* dilate with each inspiration. The loud, "sawing," stridulous breathing is present,

indicating obstruction both to inspiration and expiration. As the dyspnoea increases, all the accessory muscles of respiration are brought into action. There is now with every inspiration deep recession of the suprasternal fossa, the supraclavicular regions, and the epigastrium. The child tosses uneasily from side to side in his crib, at times struggling violently to get more air into the lungs. The pulse grows rapid and weaker. There is slight blueness of the finger nails and the lips; the face is usually pale; but later this too may be cyanotic. The skin is covered with clammy perspiration. On auscultating the chest, very rude respiratory sounds are heard, but no vesicular murmur. As the symptoms increase in severity the temperature usually rises gradually, in some very severe cases at the rate of a degree an hour, until shortly before death it reaches 104° or even 106° F. Late in the disease the intellect becomes dull, the violent struggles for air cease, and the child passes into a condition of semi-stupor which gradually deepens until death occurs, which may be preceded by convulsions.

Such is the usual course of the disease when unrelieved by treatment. Its progress is most rapid in infants, in whom death usually takes place in from thirty-six to forty-eight hours from the first symptoms. In older children the course is rather slower, and the attack may last from two days to a week, death occurring more frequently from bronchial croup or pneumonia. They are indicated by continued high temperature, rapid respiration, cyanosis, and increased prostration.

The course of the disease is not always so regular. Occasionally for a week or more the symptoms are precisely like those of catarrhal laryngitis of moderate severity—hoarseness, laryngeal cough, little or no fever, and slight or occasional dyspnoea. Then there may be the sudden development of very severe symptoms, and death in a few hours. Great improvement may follow the dislodgment of the membrane by vomiting or coughing, although in most cases it forms again.

The issue of every case of diphtheritic laryngitis is doubtful. The prognosis is worse in infants and very young children than in those over three years of age. Before the days of antitoxine the mortality of cases not operated upon was from eighty to ninety per cent. Even with modern methods of treatment the outlook in infants is bad; fully forty per cent die.

It may be difficult in a given case to decide whether the dyspnoea is due to laryngeal inflammation, and whether this inflammation is catarrhal or diphtheritic. The dyspnoea of retro-pharyngeal abscess, of foreign bodies in the larynx or trachea, or of broncho-pneumonia, may be mistaken for that due to laryngitis. But in none of these conditions should there be any doubt if a careful examination is made and a history obtained. Retro-pharyngeal abscess may be recognised by digital examination of the pharynx; broncho-pneumonia by the signs in the lungs,

the difference in the character of the dyspnœa, and especially by the absence of the noisy stridor; in the case of foreign bodies, whether they enter through the mouth or consist of ulcerating caseous glands which have ruptured into the trachea, the dyspnœa comes suddenly, and is not accompanied by fever. The main points by which catarrhal laryngitis is distinguished from the diphtheritic form have been considered under the former disease. In brief, diphtheritic inflammation may be assumed if there is severe, constant, and increasing dyspnœa with aphonia.

*Malignant Diphtheria.*—The symptoms are usually severe from the outset. The exudation in these cases may be of a yellow, dirty-gray, or olive colour, sometimes being almost black from the presence of blood. The membrane is usually extensive, covering the entire pharynx, often extending to the nose and the middle ear, and occasionally spreading to the buccal cavity. There is great swelling of the tonsils and uvula, and it is often impossible to obtain a view of the pharynx. Sometimes the inflammation is of a necrotic character, and there may be extensive sloughing of the tonsils, the uvula, or the soft palate. The nasal discharge is generally abundant, and often very offensive. There is marked swelling of the cervical lymph glands, and frequently extensive infiltration of the cellular tissue of the neck, so that the head is thrown back to relieve the pressure upon the larynx and trachea. The swelling sometimes forms a distinct collar, reaching from ear to ear and filling out the whole space beneath the jaw. The pressure upon the jugular veins leads to congestion and swelling of the face and congestion of the brain.

The temperature is usually high; it follows no regular course, but generally fluctuates widely from 102° to 106° F. In some cases, however, it may never be above 101° F. In the form characterised by very high temperature there is sometimes found a general streptococcus or pneumococcus infection, usually the former. The pulse is weak, rapid, and compressible. The peripheral circulation is poor, the extremities are often cold, there is extreme muscular prostration, and both vomiting and diarrhœa are frequent. There may be excitement, restlessness, and active delirium, or dulness, apathy, and stupor. Nephritis is very frequent and is often severe; the urine contains a large amount of albumin and casts of all varieties, but rarely blood. In a large proportion of the children under three years old broncho-pneumonia develops. Severe symptoms continue for from two days to a week; the patient may die from the sudden invasion of the larynx, or there may be suppression of urine and uræmic convulsions; but more frequently the cause of death is asthenia or broncho-pneumonia. Death usually occurs while the local disease is at its height. Occasionally it comes later from heart failure, after the signs of local improvement have begun.

Those who manage to escape the dangers of the acute period have still others to encounter. Among the latter may be mentioned, ex-

tensive sloughing in the throat or of the cellular tissue of the neck, which may be followed by severe or even fatal hæmorrhage, diffuse supuration of the same region, late nephritis, pneumonia, or pleurisy, and finally paralysis of the heart or respiration.


**Complications and Sequelæ.**—Most of the complications of diphtheria have already been mentioned either under the head of Lesions or Symptoms. It only remains to consider their clinical association.

Otitis occurs particularly in the rhino-pharyngeal cases, and is sometimes due to the diphtheria bacillus alone, but more often to mixed infection. The type of inflammation is often a severe one, and it may be accompanied by necrotic changes in the drum membrane which resemble those of scarlet fever.

Broncho-pneumonia is the most frequent complication in young children. It occurs especially in laryngeal cases, and in those of a severe type whether the larynx is involved or not. Other pulmonary complications are infrequent. Pleurisy with a serous effusion may occur in connection with severe nephritis, and empyema in septic cases. Emphysema is a complication of laryngeal diphtheria; it is nearly always vesicular, rarely interstitial. It may become general, extending into the cellular tissue of the neck and afterward that of the entire body. Pericarditis, endocarditis, and meningitis are all very rare and are seen chiefly in septic cases of the most severe type. Myocarditis is much more frequent, and is present to a greater or less degree in nearly all severe cases, although in but a small proportion of these does it give rise to distinct symptoms. It is closely connected pathologically with degeneration of the cardiac nerves, and it may be a cause of sudden death at any time during the acute period of the disease or during convalescence.

Thrombosis and embolism are among the less frequent complications. If cerebral, they may cause hemiplegia, aphasia, and sometimes convulsions; if peripheral, they usually affect one of the lower extremities, where they may cause sudden pain, numbness, and coldness of the limb, followed by partial paralysis, œdema, and sometimes even by gangrene. Thrombosis of the pulmonary artery or of the heart may be a cause of sudden death; or this may occur more gradually with dyspnoea and præcordial distress, with pallor or cyanosis. Both thrombosis and embolism are associated with a very feeble action of the heart, and generally they are preceded by degenerative changes in its muscular walls.

Hæmorrhages are usually nasal, and while in most cases they are not serious, they may necessitate plugging of the posterior nares. Bleeding from any other mucous membrane may occur, but it is rare except from the mouth. Subcutaneous hæmorrhages are infrequent, and are evidence of a very high degree of diphtheritic toxæmia. They usually occur as small petechial spots, but are sometimes extensive. They may



be seen upon almost any part of the body, most frequently upon the abdomen and lower extremities; but the most extensive extravasation I have ever seen was in the neck, reaching from the clavicle almost to the ear and covering nearly one lateral half of the neck.

Albumin is present in the urine of almost every case of moderate severity, usually depending upon acute degeneration of the kidneys. Acute nephritis is most frequently seen in severe cases. It then usually develops at the height of the local disease, but may come during convalescence. Albumin and casts are found in the urine, but rarely is there dropsy or signs of uræmia. Less frequently a more severe form of inflammation occurs, with dropsy, scanty urine, or even suppression, vomiting, and all the usual symptoms of acute uræmia. This complication may be a cause of death.

Functional disturbances of the stomach are present in most of the severe cases, but lesions of the mucous membrane are rare. While diarrhœa is often seen without intestinal lesions, the latter are of frequent occurrence. The most characteristic form of inflammation is a follicular ileo-colitis, which, however, seldom goes on to ulceration. It is extremely rare that the membranous form is seen, and then it is almost always associated with the presence of other organisms than the diphtheria bacillus.

Diphtheria is usually followed by a severe and often persistent anæmia which may continue for weeks. Pneumonia, nephritis, and cardiac disease may first show themselves during convalescence, and so be ranked as sequelæ. The most important sequel of diphtheria, however, is post-diphtheritic paralysis, already discussed in the chapter on Multiple Neuritis.

*Pneumogastric Paralysis.*—Some cases of diphtheria, especially those which receive no antitoxine or when the antitoxine is administered late or in too small amount, present a group of symptoms which have been referred to degeneration of the pneumogastric nerves. The evidence, however, is by no means conclusive that this is the true explanation of the clinical picture, which is a familiar one.

These symptoms may come on at any time in the course of the disease, but seldom earlier than the end of the second week. By this time the throat has usually cleared off entirely, and the patient is considered convalescent. The symptoms relate to the stomach, the heart, and the respiration. Usually the first thing to attract notice is that the patient refuses food and vomits occasionally, afterward persistently, without apparent cause. If the pulse is carefully observed it is found to be much slower than previously, being only 80 or 90 when it was formerly 120 or more. It is also weaker, compressible, and often somewhat irregular. The face is pale or slightly cyanotic, and moderate dyspnœa may be noticed. There are frequent attacks of severe abdom-

inal pain which comes in paroxysms, and is usually referred to the epigastrium. These symptoms in most cases gradually increase in severity for two or three days, but sometimes develop with such intensity that death occurs within twelve or twenty-four hours. The later symptoms are a continuance of the abdominal pain and vomiting; there is a feeling of great precordial oppression and distress accompanied by dyspnoea; the respiration is shallow and often rapid; the face is either pale or cyanotic; the extremities, cold; the pulse, slow, irregular, and intermittent, becoming rapid on the slightest exertion. The heart sounds are weak, the muscular quality is absent, and the rhythm much disturbed. There may be no murmurs. There is great restlessness, but the mind is entirely clear. Death usually results from heart failure, which may come quite suddenly, often from so slight exertion as turning over in bed or attempting to take food.

Not all the cases are so severe. In the milder forms there is some palpitation, an irregular pulse, slight dyspnoea, and occasional syncopal attacks, but of no great severity. Such symptoms may come and go for several days and then disappear; but more frequently they prove to be the beginning of the more serious form of the complication. The time of occurrence of these symptoms varies considerably. It may be as late as the third or fourth week. The late cases are generally associated with some other form of post-diphtheritic paralysis.

Sudden heart failure may be seen late in diphtheria quite apart from the symptoms just described. It may occur with few or no premonitory symptoms; as when a child falls dead after walking across a room, or suddenly sitting up in bed, or from some other muscular effort, or possibly as a consequence of passion or excitement. I knew of one little girl who was considered well enough to go coasting and who died suddenly after the effort.

The explanation of heart failure during or after diphtheria is therefore not always the same. When it occurs at the height of the disease it is sometimes due to cardiac thrombosis, probably always associated with changes in the muscular walls. When it occurs late and follows some sudden muscular effort or excitement without premonitory symptoms of any sort, it is probably the result of changes in the muscular walls—a toxic myocarditis.

**Diagnosis.**—The diagnosis of diphtheria rests upon two kinds of evidence—clinical and bacteriological. In mild cases and in the early stage only bacteriological evidence can be relied upon. However, the clinical manifestations of the disease are important and should not be ignored. It is in most cases possible to say from clinical symptoms that a case is one of diphtheria; but it is never possible to say from symptoms alone that a case is not diphtheria. Cultures, therefore, are of the greatest assistance, and should if possible be made in every case. They are nec-

essary in the mild cases in order that a correct diagnosis may be made and proper quarantine regulations enforced; otherwise a case might be dismissed as simple tonsillitis and no precautions taken.

The mere presence of diphtheria bacilli in the throat does not prove that a person has diphtheria any more than the presence of the pneumococcus in his saliva proves that he has pneumonia; but when diphtheria bacilli are associated with clinical evidences of inflammation of the throat or nose the diagnosis may be regarded as established. Again, the case may be one of diphtheria and the bacilli not found at the first examination, although found subsequently. In using antitoxine one must, in perhaps the majority of cases, be guided by clinical symptoms alone, not waiting for the result of the bacteriological examination. It is therefore important that both methods of diagnosis should be employed.

1. THE CLINICAL DIAGNOSIS.—Not much importance can be attached to the mode of onset; for diphtheria may begin in many different ways. The presence of a nasal discharge, especially if abundant, ichorous and tinged with blood, the early development of the symptoms of croup, the rapid enlargement of the cervical lymph glands, and the early appearance of albumin in the urine—all point strongly to diphtheria. Later symptoms which are especially diagnostic are marked anæmia, progressive asthenia, intense toxæmia often with a low temperature, very feeble pulse which is sometimes slow, sometimes rapid, sudden attacks of syncope, nasal hæmorrhages, nasal regurgitation from paralysis of the soft palate, contagion, and, finally, the development of paralysis of the muscles of the throat, eye, or extremities, with paralysis of the heart or respiration.

The membrane of diphtheria generally appears first upon the tonsils, usually as a gray film which gradually becomes more dense and white, and often has the look of being plastered on. The colour of older membrane is gray, greenish-yellow, brown, sometimes black. Beginning as a small patch, it soon covers the tonsils. It frequently affects one tonsil twenty-four or thirty-six hours before the other, and occasionally it is confined to one side. In exceptional cases it begins in the crypts of the tonsil and appears as isolated dots, which may coalesce to form a continuous patch like that already described, or it may remain isolated like the exudate of an ordinary follicular tonsillitis. More important is the fact that the membrane spreads from the original seat, and also the manner of its spreading. If it extends beyond the tonsils to the walls of the pharynx, the faucial pillars, and the uvula, it is almost surely diphtheria. The same is true of doubtful patches on the tonsils or fauces followed by symptoms of croup. The rapidity of the spreading varies much in the different cases, depending upon the intensity of the infection; but the gradual extension, as shown by observations made at in-



tervals of six or eight hours, usually settles the diagnosis in the primary cases. However, if the throat symptoms complicate measles or scarlet fever the above rules do not apply. Most of the membranous inflammations of the throat seen in these diseases are not due to diphtheria. This is particularly true of those which occur at the height of the primary disease. Those which develop at a later period are often due to diphtheria.

Primary membranous inflammation of the larynx may always be safely regarded as diphtheria; but if there is no visible membrane, the diagnosis is rendered positive only by a bacteriological examination. This may be true of many nasal cases where the only symptoms are a discharge of the character previously described. Such cases may continue for weeks with no symptoms other than the discharge, especially in infants.

It is seldom difficult to distinguish diphtheria from any other disease; but the exudation upon the pharynx or tonsils may be confounded with thrush or ulcero-membranous angina. The appearance of the tonsils on the second or third day after tonsillotomy has been performed, may easily be mistaken for diphtheria by one who is unfamiliar with the appearance of the post-operative wound.

Diphtheria of the mouth may be mistaken for herpetic or ulcerative stomatitis; but, as a rule, it is seen only in the worst cases of pharyngeal diphtheria. Diphtheria of the mouth alone is so rare that it may be ignored.

It is sometimes difficult to distinguish cases of scarlet fever in which the throat symptoms are severe and appear early, from cases of primary diphtheria. In many of these cases the eruption appears late, and is not characteristic. Much importance is to be attached, as pointing toward scarlet fever, to a prevailing epidemic, a history of exposure, a sudden onset with severe symptoms, vomiting, prostration, very high temperature, and to a very active inflammation in the pharynx. In all cases with a sudden onset, in which from the throat symptoms one is inclined to make a diagnosis of diphtheria, the possibility of scarlet fever should not be forgotten, and one should never omit to examine the patient thoroughly for an eruption.

2. THE BACTERIOLOGICAL DIAGNOSIS.—*The Technique.*—In many cases an immediate diagnosis may be reached by the examination of a cover-glass smear from the throat. This method, although often valuable, is not adapted for general use, as bacilli directly from the throat are much less typical than those from cultures, and the chances of contamination are much increased. Furthermore, the mouth often contains other bacilli which somewhat resemble the diphtheria bacillus.

In taking a culture from the throat, the tongue should be depressed and the tonsils, pharynx, or other seat of visible membrane rubbed firmly

with a swab, which is then rubbed over the surface of the culture-medium in the tube or on the plate. In laryngeal cases the culture should be taken from the posterior wall of the pharynx, and in nasal cases from the nostril. The tube or plate is then placed in an incubator for eight to twelve hours, at the end of which time the colonies may be examined. Examination, in the great majority of cases, shows either a great number of diphtheria bacilli and a few cocci, or only cocci in pairs or short chains; exceptionally, the cocci and bacilli may be present in nearly equal numbers. A definite opinion should not be given without examining several colonies.

*The Reliance to be Placed upon Bacteriological Diagnosis.*—The diphtheria bacillus will almost invariably be found, if there is visible membrane in the pharynx, if no antiseptics have been applied shortly before using the swab, and if the culture has been made with sufficient care to avoid contamination.

The diphtheria bacillus sometimes disappears early; hence cultures made while the membrane is loosening may be negative. If the membrane has disappeared, or if none has been present, it is not infrequently necessary to go into the tonsillar crypts with a probe or spoon to discover bacilli. It is therefore important in all cases to consider the duration of the disease before drawing a conclusion from a negative culture. If the case is one of laryngeal disease without pharyngeal exudation, an early culture is negative in nearly half the cases; although a little later bacilli may be coughed up and found in the pharynx in abundance. A single negative culture should never be taken as conclusive.

For diagnostic purposes, all bacilli present in suspicious throats, having the morphological and cultural characteristics of diphtheria bacilli, are to be regarded as virulent.

*Non-virulent Bacilli Resembling the Diphtheria Bacillus.*—There may be found in throats a form which corresponds in every other characteristic with the diphtheria bacillus, but which lacks virulence, as shown by animal tests. Also, another form, which, though in many particulars resembling the diphtheria bacillus, differs from it in being shorter, plumper, and more uniform in size, and in producing an alkali in broth cultures; to this the term *pseudo-diphtheria bacillus* has been given. It is more frequently seen than the form just described and like it is non-virulent. Both these forms are rare in throats where a suspicion of diphtheria exists.

*The Presence of Virulent Bacilli in the Throats of Healthy Persons.*—That virulent bacilli may be harboured for an indefinite period in the throat or nose of a healthy person is proved by many observations. The New York Health Department made observations upon forty-eight children in fourteen families in which one or more cases of diphtheria had occurred, and where no attempt at isolation had been made. In one-

half these cases bacilli were found, and animal tests showed them to be virulent in every one of six cases tested, although four of the children did not develop diphtheria. Of the entire number, forty per cent subsequently developed diphtheria. My own experience in two institutions where diphtheria has been endemic, fully confirms the observation that bacilli of all degrees of virulence are very frequently found in the noses or throats of such exposed children, although a large proportion of them never develop the disease. Outside of institutions and infected tenement houses, however, such a condition is extremely rare.

**Prognosis.**—Many possibilities exist, and even the mildest case must be regarded as serious and carefully watched, since one can never know when unfavourable symptoms may develop.

The factors to be considered in the prognosis of any given case are: the age and previous condition of the patient; the extent of the membrane and the rapidity with which it is spreading; the degree of diphtheritic toxæmia as shown by the condition of the pulse and the nervous symptoms; whether or not the membrane has invaded the larynx; and the presence or absence of complications, especially nephritis and broncho-pneumonia; but of more importance than any or all these things is whether antitoxine is used and when it is administered.

The following figures are from the Report of the Health Department of Chicago of cases treated from October 5, 1895, to February 28, 1899:

|                       |       | Died. | Mortality.     |
|-----------------------|-------|-------|----------------|
| Injected 1st day..... | 355   | 1     | 0.27 per cent. |
| “ 2d day.....         | 1,018 | 17    | 1.67 “         |
| “ 3d day.....         | 1,509 | 57    | 3.77 “         |
| “ 4th day.....        | 720   | 82    | 11.39 “        |
| “ later.....          | 469   | 119   | 25.37 “        |
| Totals.....           | 4,071 | 276   | 6.77 “         |

In all these cases the diagnosis of diphtheria was confirmed by cultures.

Diphtheria mortality is highest during the first two years of life, from its strong tendency to invade the larynx and lower air passages, and from the frequency with which broncho-pneumonia occurs as a complication. Those whose experience with this disease does not antedate the introduction of antitoxine can scarcely appreciate the results previously obtained. Of eighty-five consecutive cases under twenty-six months of age observed in the New York Infant Asylum, in a period extending over two years, the mortality was sixty-eight per cent; in over two-thirds of the fatal cases the disease involved the larynx. In diphtheria hospitals, where most of the mild cases included in the above statistics would probably not have been admitted, the mortality in children under two years formerly varied from sixty to eighty per

cent; in private practice it ranged for this age from thirty to sixty per cent.

It can not be too often emphasised that the danger from diphtheria is not over when the throat has cleared. The most frequent causes of death after this time are broncho-pneumonia and cardiac paralysis.

**Prophylaxis.**—In no infectious disease, smallpox alone excepted, can so much be accomplished in the way of prevention as in diphtheria.

Public funerals of children dying from diphtheria should invariably be prohibited. Schools should be closed whenever the disease is epidemic. Children from families where diphtheria exists should not be allowed to attend school, nor mingle in any way with other children, for the reasons that they may, while healthy, be the carriers of the disease; and, what is even more important, that they may be themselves suffering from diphtheria in an early stage or in a mild form.

In every large city, hospitals for diphtheria patients should be established, not only for the poor, but with private rooms for cases developing in hotels or other places where isolation is impossible. Every city should be provided with a steam disinfecting plant, where carpets, blankets, bedding, etc., can be sent from the sick-room for disinfection.

**Quarantine.**—Not only every undoubted case of diphtheria, but every suspected case, should be immediately isolated. Quarantine for the latter should continue until the diagnosis is settled either by a bacteriological examination or by the course of the disease. Positive and suspected cases should not be isolated together. The quarantine in every instance must be complete. If possible, cultures should be taken from the throats of all exposed children. Those containing diphtheria bacilli should be quarantined like cases of diphtheria, for they may be equally dangerous; they should use gargles and sprays, and the nose and throat should be closely watched.

Bacteriology has furnished some very definite data from which the necessary duration of the period of quarantine may be determined. In this the physician is to be guided by the time that the bacilli remain in the throat, for the patient is to be considered as dangerous while they persist. This point was investigated by the New York Health Department in 605 cases: In 304 of these the bacilli had disappeared by the third day after the membrane was gone; and in 301 they persisted for a longer time—in 176, for seven days; in 64, for twelve days; in 36, for fifteen days; in 12, for twenty-one days; in 4, for twenty-eight days; in 4, for thirty-five days; and in 2, for sixty-three days. Many of the cases in which the bacilli have persisted for an unusual time have been those of nasal diphtheria; in some of these it is doubtless owing to the fact that the nasal sinuses, especially the antrum, have been invaded. While it is unquestionably true that in a certain number of cases these persistent bacilli are non-virulent, the opposite has been frequently shown.

Of 15 cases in which the virulence was tested, virulent bacilli were found in 9 at periods varying from eight to twenty-five days after the membrane was gone.

*Treatment of Suspected Cases.*—During an epidemic of diphtheria, especially in an institution, every sore throat and nasal discharge should be looked upon with suspicion, and isolated pending the result of a bacteriological examination, even though no membrane is present. If there are patches on the tonsils or any other visible membrane, the case should be treated as true diphtheria, in order that no time may be lost. If the bacteriological examination shows the disease not to be true diphtheria, the patient may be released from quarantine in two or three days, provided the throat symptoms disappear. It is, of course, important that the conditions laid down with reference to bacteriological diagnosis shall have been fulfilled. Should symptoms continue, however, a second culture should be taken.

*Immunisation of Persons Exposed.*—When a case of diphtheria occurs in a family or an institution, every child that has been exposed should receive an immunising dose of antitoxine. This rule is not followed in practice as regularly as it ought to be. There is no doubt that for a limited time—from two to three weeks—the serum confers almost complete protection.

One need not hesitate to immunise persons of any age and in almost every condition, even newly-born infants and pregnant women.

The dose for immunisation is from 500 to 1,000 units, the former being that required for an infant, and the latter for older children. If the exposure is continuous, as in an institution, the dose should be repeated every three or four weeks. A nurse in charge of a diphtheria case should receive 1,000 units.

Diphtheria so often complicates scarlet fever and measles, particularly in institutions and in hospitals for contagious diseases, that special consideration should be given to such patients. It is practically impossible by cultures to separate with absolute certainty all cases in which diphtheritic infection is present, from others; the only safe rule is to immunise every child admitted to a scarlet-fever or measles hospital, and in institution epidemics of either of these diseases to immunise every child attacked.

*Nurses and Physicians.*—As diphtheria is contracted, not from the breath of the patient or the air of the room, but by receiving the bacilli into the mouth or air passages, all possible means should be taken to destroy the bacilli discharged, and to secure absolute cleanliness in everything about the sick-room. When it can possibly be avoided, nurses should not be allowed to eat or sleep in the sick-room, and an antiseptic gargle should be used. The hands should be kept clean, and only such dresses worn as can be readily washed and disinfected. It is the nurse

who is most likely to contract the disease, on account of the continued exposure.

The physician should take the same precautions as in scarlet fever. A pocket tongue-depressor should not be used for the throat, but a wooden depressor or a spoon kept in a solution of carbolic acid.

*The Sick-room.*—The carpets, hangings, upholstered furniture, everything in fact not necessary for the patient's welfare, should be removed. The room should be a large one, well ventilated, and fresh air should be allowed in abundance. The floor should be washed once a day with a solution of bichloride, 1 to 2,000, and dusted often with cloths moistened in the same solution. All handkerchiefs, bed-linen, and clothing removed from the patient should be treated as in a case of scarlet fever. Pieces of membrane and other matters discharged from the patient should be burned. Old muslin or absorbent cotton should be used to cleanse the nose and mouth of the patient and burned immediately. All vessels for the reception of expectoration or other discharges should contain bichloride, 1 to 2,000. The bed-linen should be very frequently changed, and everything kept scrupulously clean. In the room should be a large bowl of carbolic acid, 1 to 40, or some similar solution for cleansing the hands, and a tray of the carbolic solution for spoons, syringes, or other things used in the treatment of the patient. All spoons, cups, or other dishes used by the patient should be carefully sterilised by boiling. No milk or other food should be allowed to stand about the room. There is no objection to the hanging of sheets moistened in carbolic, bichloride, or other disinfectant solutions before the door, but neither this nor hanging them about in the sick-room is to be regarded as having any value in disinfecting the air of the room. They create a false sense of security, and often lead to the neglect of thorough cleanliness.

Disinfection of apartments after an attack should be done as after scarlet fever.

**Treatment.**—*General Measures.*—It is important in every case that there should be plenty of fresh air in the room throughout the attack. Hospital patients should never have less than 1,000 cubic feet of air space, and if possible 1,200 should be allowed. Even in mild cases the patient should be kept in bed throughout the entire attack, and in severe cases this should be continued for some time during convalescence.

Nursing infants may be fed on breast-milk obtained by a breast-pump, but should not be put to the mother's breast. The feeding of older children should be managed very much as in other cases of severe illness. Milk is the main reliance; it should usually be diluted. The greatest difficulty in feeding is seen in the latter part of the disease, when the patients are septic and have a strong aversion to food, when vomiting is easily excited and when swallowing is difficult on account of the swelling and pain. It is then that gavage is most valuable. This is much

more successful with children under three years old than is rectal feeding. In older children the tube may be passed through the nose.

*Stimulants.*—In most cases they are not needed until the third or fourth day, and in some they may not be required at all. The indications for stimulants are marked prostration, a feeble pulse, and a weak first sound of the heart. Of alcohol, half an ounce of whisky or brandy in twenty-four hours is enough to begin with, for a child four years old. This should be diluted with at least eight parts of water. In very severe cases two or three times as much may be given; but more than this, except for a short period, is seldom wise. More reliance is to be placed upon the other circulatory stimulants, especially caffeine, camphor, and digitalis, which are given for the same indications as in other acute diseases. In cases of threatened cardiac paralysis occurring late in the disease or during convalescence, morphine should be used hypodermically. Full doses must be given and repeated every two to four hours, so that the child may be kept under its influence.

Except for stimulation or the control of special symptoms such as vomiting or diarrhoea, all internal medication should be omitted; for there is yet wanting proof that drugs influence the course or the result of the disease.

*Local Treatment.*—Since the introduction of antitoxine, opinion has undergone a decided change with reference to local treatment. While it should not be entirely abandoned, still it is of secondary importance; and under conditions when it can be carried out only with great difficulty and the use of force it is often wise not to attempt it regularly.

The purpose of local treatment, it is now generally agreed, should be cleanliness, and not the destruction of bacilli. Cleanliness of the nose, mouth, and pharynx is important, inasmuch as one of the chief dangers of the disease is the aspiration of bacteria contained in the abundant secretions of these parts, into the larynx and bronchi. Our aim should therefore be to keep the parts as clean as possible without too severely taxing the strength of the child.

For cleansing the nose and pharynx only syringing can be depended upon. Nasal syringing is indicated when there is much nasal discharge, whether membrane is visible in the anterior nares or not. In septic cases with a profuse foetid discharge it may be necessary to syringe the nose, no matter how strongly the child resists. Whether it shall be done, will depend upon the condition of the patient's strength and his pulse. The purpose in syringing is not so much to clear the nose, from which absorption is slow and imperfect, as to flush the rhino-pharynx, from which absorption is always very active. Only bland solutions should be employed, such as a saline solution, one per cent, or a boric-acid solution, one- to four-per-cent strength. For some cases, the piston syringe may be used; but for most a fountain syringe possesses man-

ifest advantages, and it is rather more convenient for hospital purposes. Irrigation of the pharynx is best done with the fountain syringe, and is of especial value where there is much swelling or abundant discharge. All solutions should be used as warm as can be borne, and in sufficient quantity to irrigate the parts thoroughly, a few such irrigations being much better than a great many partial ones. By a skilful nurse syringing can in most cases be done with comparatively little disturbance to the child.

Slight nasal hæmorrhages may necessitate less frequent syringing, and a free hæmorrhage may require it to be discontinued. Astringent solutions of alum and adrenalin are often beneficial in such cases, but they must be used carefully. In children who are old enough gargles should be used. A solution of boric acid, or Dobell's or Seiler's solution much diluted, may be employed.

In cases with a moderate nasal discharge it is usually sufficient to syringe three or four times a day; but in severe septic cases, with very abundant discharge, syringing should be repeated as often as every two hours during the day and every four hours at night.

External applications to the throat have practically no effect upon the disease, but are often useful to relieve pain and tension in the swollen lymph-glands. Poultices should not be employed. As a continuous application, only cold is to be advised, generally by means of an ice-bag well protected to prevent wetting the clothing.

The treatment of cardiac and other forms of post-diphtheritic paralysis has been considered in the chapter on Multiple Neuritis.

**The Serum Treatment.**—This has been the outcome of a long series of experiments in which many men have had a share; but it is to Behring pre-eminently that the credit belongs for the development of the principles of serum-therapy.

Antitoxine is produced by the cells of the body under the stimulus of the diphtheria toxine. It is intimately combined with the globulin of the blood, and is itself possibly a globulin. It directly neutralises the toxine produced by the diphtheria bacillus, and also has some effect upon the bacilli themselves, the nature of which is not understood. It induces a condition in the blood which inhibits the growth of the bacilli, and thus arrests the membranous inflammation which they excite.

Properly prepared, it will keep without deterioration for from three to six months; but after one year it loses somewhat its antitoxic properties. It should be kept in a cool, dark place, and after a bottle has been opened it should be used within a few days. Antitoxine is now prepared in a dry form, which is to be preferred only when it must be kept for a very long time.

The strength of the serum is measured in antitoxine units, the unit being an arbitrary one, viz., the amount of antitoxine which will protect



a guinea-pig weighing 250 to 300 grammes against one hundred times the fatal dose of diphtheria toxine. The improvements in the production of the serum have thus far consisted in increasing its strength. Behring's serum first used contained but one unit in each cubic centimetre. At present there can be obtained sera containing 1,000 antitoxine units in each cubic centimetre. This concentration is of immense advantage and has to a large degree done away with the unpleasant symptoms.

*Method of Administration and Dosage.*—Before making the injection, the skin should be thoroughly cleansed with alcohol; the needle should invariably be boiled and the whole syringe either boiled or rinsed with alcohol. The seat of injection is not a matter of great importance; my own preference is for the cellular tissue of the abdomen or the muscles of the buttock. Absorption from the cellular tissue is slower than from the muscles. For very rapid effect, intravenous injections should be employed. After the injection is made the puncture should be covered by adhesive plaster.

It is desirable to give enough antitoxine to neutralise the diphtheria toxine present in the blood, but no amount can neutralise the toxine which has already become fixed to the cells, except to a very slight degree. What can be accomplished is to supply the blood with sufficient antitoxine to neutralise new toxine as fast as it is produced. Convinced now of the essential harmlessness of the serum, the tendency everywhere has been to use larger and larger doses. For a child over two years old an initial dose for a severe attack, including all laryngeal cases, should not be less than 7,000 or 8,000 units, repeated in from six to eight hours, provided no improvement is seen. Children under two years should receive from 5,000 to 6,000 units. Cases of exceptional severity, in older children, should receive from 10,000 to 15,000 units, to be repeated in from six to eight hours if the progress of the disease is unfavourable. Mild cases should receive from 3,000 to 5,000 units as an initial dose, a second being rarely required.

In cases receiving antitoxine late, even though the symptoms may not seem particularly severe, the dose should be increased in proportion to the length of the illness—i. e., if three days ill, three times the ordinary dose should be given.

Only serum from a trustworthy manufacturer should ever be used. The most concentrated serum which can be obtained should be selected.

All experience shows that the results are greatly modified by the time of its administration. The serum can not undo the serious damage already done to the cells of the body, and this at the time of injection may be so great that death will result. In very mild cases, with older children, one may wait for the result of a bacteriological examination, but never in a severe case and never in a young child. In the group of

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severe cases should be placed every one which at the first visit shows a pharyngeal exudate covering more than the tonsils, also all cases with symptoms of laryngeal invasion, and all with an exudate on the pharynx and a profuse nasal discharge. If in a doubtful case twelve hours' observation shows that the membrane has spread from its original seat, no further delay is admissible. In human diphtheria marked benefit usually follows injections made as late as the third day; but after this time the value of the serum diminishes very rapidly, and although striking examples of benefit are sometimes seen after later injections, they can not be depended upon. In very severe or in malignant cases so much harm may be done during the first twenty-four hours of the attack that the subsequent use of antitoxine is without avail.

*The effect upon the diphtheritic membrane* is usually noticeable within twenty-four and often in twelve hours; it first stops spreading, and soon begins to soften and loosen. The swelling of the mucous membrane subsides and the local disease abates, very much as when the disease runs its usual course. The striking thing after the use of antitoxine is the rapidity with which these changes take place, and the abrupt transition from an advancing to a retrograde process. The subsidence of the inflammatory conditions in the larynx and trachea is quite as marked as in the pharynx. The symptoms of stenosis, even when severe, often diminish in a few hours, making operation unnecessary in a very large number of cases when previously it seemed inevitable. The membrane loosens rapidly in the larynx and trachea, sometimes necessitating the frequent removal of the intubation tube, when operation has been performed. Improvement is also shown by the cessation of the nasal discharge, the re-establishment of nasal respiration, and the diminution in the swelling of the glands of the neck.

The effect upon the constitutional symptoms is not less striking. In favourable cases there is seen, often in twelve hours, a fall in temperature and improvement in the pulse and in the nervous symptoms.

*The Limitations of Antitoxine.*—It is important that these should always be kept in mind. The serum must be given early, for if given late it can not undo the mischief already done by the diphtheria toxine. Cases of great severity often pass the period when recovery is possible, before the antitoxine is given. This period may in some cases be four days, in others it may be less than twenty-four hours. The tissues most susceptible to the diphtheria toxine are probably those of the nervous system, the heart, and the kidneys; and the consequences of its action may be seen in the production of nephritis, in heart failure at the height of the disease, or in later paralysis of the heart, respiration, or the voluntary muscles, in spite of the fact that antitoxine is given at a period early enough to avert death from local disease in the larynx or bronchi. Against the phlegmonous inflammation of the throat or the cellular

tissue of the neck, broncho-pneumonia, and nephritis, antitoxine is powerless; and just in proportion to the severity of these inflammations are negative results seen.

*Eruptions and Other Unpleasant Effects.*—Some transient, local œdema usually follows the injection and a slight rise of temperature is very frequently observed. In a few hours there may be seen a general erythema; this, however, is rare and usually of short duration. The most important eruptions are seen between the eighth and fourteenth days. They follow from ten to twenty per cent of the injections made, and appear to be quite independent of the amount of serum used. The exact cause is not known. The most common eruption is urticaria. This is often intense, very annoying, and may nearly cover the body. It may be accompanied by a slight rise of temperature; it usually lasts for two or three days; however, it is rarely severe for more than twenty-four hours. Various forms of erythema are occasionally met with. In two or three instances I have seen hæmorrhagic eruptions, generally in the neighbourhood of the large joints, and always in children suffering from extreme malnutrition. In a few cases a moderate swelling of some of the joints has been observed, and very exceptionally a transient albuminuria. One occasionally meets with patients who seem unusually susceptible to serum injections, and in whom even small immunising doses cause headache, muscular pains, and general malaise, so that they feel quite wretched for several days. All of the above symptoms except the urticaria are rare, and should not for an instant deter one from using antitoxine when indicated.

*Real and Alleged Dangers from Antitoxine Injections.*—In a few instances sudden death has followed antitoxine injections, but the evidence that antitoxine was the cause of death has not always been conclusive. In some of these patients the autopsy has revealed a status lymphaticus not before suspected. In this condition the shock of so slight a thing as a needle puncture may produce death. There are other cases which do not admit of this explanation. Almost all have occurred in patients during adolescence or adult life. So many of these patients have been asthmatics that the association can hardly be an accidental one; and curiously some of these patients have had the form of asthma excited by contact with horses. The symptoms usually come on within a few minutes after the injection, and occur quite independently of the dose given. Several have followed small immunising doses given to apparently healthy persons. The most striking symptoms are a rapidly developing dyspnœa with cyanosis and great prostration. In the most severe cases death may follow in a few minutes from suffocation; in those less severe, a gradual recovery takes place with no permanent after-effects. The most effective treatment is atropine, hypodermically, in full doses, combined with artificial respiration.

That so very few reported instances of serious harmful results have occurred among the great numbers of injections which have been made, is sufficient to establish the fact that the serum itself is, in the vast majority of instances, quite harmless. Certainly in children one should not hesitate one moment in regard to its use. In an asthmatic patient, if antitoxine is given, atropine should be injected simultaneously.

*Results with Antitoxine Treatment.*—Since 1895 the serum has been tested on such an extensive scale as the prevalence of diphtheria all over the world has made possible, with results so uniformly good that it seems quite unnecessary any longer to cite statistics in proof of the value of this remedy. No tables of figures are so convincing to an individual as personal experience, and by this argument one by one the opponents of antitoxine have been converted.

The beneficial effects of the remedy may be summed up in the following statements: (1) The percentage mortality from diphtheria in hospitals both in Europe and in America has been reduced to a little more than one-third the previous figures; (2) the proportion of cases now requiring operation for laryngeal stenosis has been reduced to about one-half; (3) the mortality after tracheotomy has been reduced to one-half, and that after intubation to about one-third the former figures; (4) but even more convincing is the effect of the serum treatment upon the actual diphtheria mortality of cities and countries where it has been used.

In the first of the subjoined tables is given for a period of years the actual number of reported deaths from diphtheria and membranous croup, irrespective of the growth in population; in the second one the number of deaths in each 10,000 of population. These figures can not be open to the question which is sometimes raised concerning percentage mortality statistics.

*Table Showing Annual Deaths from Diphtheria and Croup, before and since the Use of Antitoxine.*

|   | 1887   | 1888   | 1889   | 1890   | 1891   | 1892   | 1893   | 1894   | 1895  | 1896  | 1897  | 1898  | 1899  | 1900  |
|---|--------|--------|--------|--------|--------|--------|--------|--------|-------|-------|-------|-------|-------|-------|
| London . . . . .                                | 1,579  | 1,812  | 2,075  | 1,877  | 1,174  | 2,182  | 3,484  | 2,861  | 2,479 | 2,793 | 2,328 | 1,842 | 2,041 | 1,558 |
| Berlin . . . . .                                | 1,392  | 1,195  | 1,210  | 1,601  | 1,106  | 1,342  | 1,637  | 1,416  | 987   | 559   | 546   | 664   | 655   | 563   |
| Paris . . . . .                                 | 1,585  | 1,729  | 1,706  | 1,659  | 1,361  | 1,403  | 1,206  | 1,009  | 435   | 444   | 268   | 256   | 336   | 291   |
| New York . . . . .<br>(Manhattan and<br>Bronx)  | 3,050  | 2,553  | 2,291  | 1,783  | 1,970  | 2,106  | 2,558  | 2,870  | 1,976 | 1,763 | 1,591 | 843   | 960   | 1,121 |
| Chicago . . . . .                               | 1,405  | 1,297  | 1,509  | 1,261  | 1,358  | 1,548  | 1,476  | 1,460  | 1,632 | 1,098 | 774   | 680   | 917   | 797   |
| Boston . . . . .                                | 410    | 589    | 683    | 462    | 285    | 481    | 546    | 878    | 654   | 372   | 456   | 185   | 304   | 537   |
| Philadelphia . . . . .                          | 858    | 523    | 727    | 748    | 1,362  | 1,707  | 1,238  | 1,452  | 1,398 | 1,201 | 1,514 | 1,154 | 997   | 1,064 |
| Brooklyn . . . . .                              | 1,453  | 1,885  | 1,467  | 1,283  | 1,180  | 1,137  | 878    | 1,660  | 1,454 | 1,310 | 998   | 745   | 744   | 673   |
| Denver . . . . .                                | 68     | 120    | 109    | 277    | 175    | 89     | 106    | 71     | 40    | 19    | 43    | 34    | 31    | 14    |
| Germany . . . . .<br>(966 towns over<br>15,000) | 10,970 | 10,142 | 11,919 | 11,915 | 10,484 | 12,365 | 16,557 | 13,790 | 7,611 | 6,262 | 5,208 | 5,220 | 5,111 | 4,685 |
| N. Y. State . . . . .                           | 6,490  | 6,710  | 5,030  | 4,954  | 4,844  | 5,970  | 5,942  | 6,616  | 5,696 | 4,640 | 4,115 | 2,612 | 2,786 | 3,306 |
| Mass. . . . .                                   | 1,628  | 1,831  | 2,214  | 1,626  | 1,218  | 1,455  | 1,394  | 1,801  | 1,784 | 1,677 | 1,426 | 706   | 1,047 | 1,475 |

<sup>1</sup> Cases reported 1899, 7,134.

<sup>2</sup> Cases reported 1900, 12,041.

*Table Showing Average Annual Deaths from Diphtheria and Croup per 10,000 of Population.*

|   |  |
|---|--|
| London, before antitoxine, 1887-'93, 4.8; since antitoxine 1896-1900, 4.7 |  |
| Berlin, " " 10.2; " " 3.7   |  |
| Paris, " " 6.5; " " 1.3   |  |
| New York, " " 14.5; " " 6.3   |  |
| Chicago, " " 13.1; " " 5.0  |  |
| Denver, " " 12.9; " " 1.7   |  |
| Philadelphia, " 1890-'94, 11.9; " " 9.6                                   |  |

Some explanation of these figures is necessary that they may be fully appreciated. The great reduction in the death-rate is seen only in those cities and countries where the serum treatment has been widely employed. Nowhere in Europe is this true to the same degree as in Paris, Berlin, and Germany generally; and probably nowhere in Europe was it so little used and so slow in gaining favour as in London. In our American cities the effect of the serum treatment upon municipal mortality figures has been directly proportionate to the extent to which the health departments have believed in its efficacy and encouraged its use by furnishing it free to the poor, and sending their own inspectors to administer it. This is true particularly of New York and Chicago; in Philadelphia, on the contrary, the authorities for a long time were openly opposed to the serum treatment.

*Convalescence.*—After a severe attack of diphtheria convalescence is always slow on account of the anæmia and the depressing effects of the disease. Patients should invariably be kept in bed for at least a week after the throat has cleared, and longer if any tendency to cardiac weakness is seen. The pulse should be carefully watched, and irregularity, intermission, dicrotism, or a weak first sound of the heart, should make one apprehensive. An abnormally slow pulse is generally more serious than one which is rapid. Under such circumstances the patient should be kept recumbent and absolutely quiet, since sudden and even fatal syncope may be the result of a violation of these rules.

The extreme degree of anæmia requires that iron be given for a considerable time during convalescence, to be followed by cod-liver oil and other tonics.

Great difficulty is occasionally experienced in getting rid of the bacilli in the throat. The tonsillar crypts, the adenoid tissue of the rhino-pharynx, and the nasal sinuses are the places where the bacilli are most likely to remain. Inasmuch as it is now generally made a condition of release from quarantine that the throat shall have been shown by cultures to be free from bacilli, this becomes a matter of much importance. The most efficient means appears to be to syringe the nose gently three or four times daily with a solution of bichloride, 1 to 10,000, to which one-eighth glycerin has been added, and to use the same solution

as a gargle. For children under four years old a simple salt solution, or a dilute Dobell's solution, should be substituted and the gargle omitted.

*Laryngeal Diphtheria.*—Emetics, inhalations of steam, and solvents for the membrane, although they all sometimes give relief, are not to be relied upon.

Opinions will always differ as to the time when operative interference is called for. One should never wait for general cyanosis, for often this does not occur until just before death. It is better to operate too early than too late. If, in spite of other measures, the dyspnoea increases steadily, operation should not be deferred longer. Intubation has almost universally superseded tracheotomy as a primary operation for the relief of membranous laryngitis. Tracheotomy is still needed at times for the cases, very few in number, in which intubation fails to give relief on account of the position of the membrane or some other complication.

#### *Intubation.*

Intubation is the introduction of a tube through the mouth into the larynx for the relief of laryngeal dyspnoea. For the operation, as now performed, the world is indebted to the late Dr. Joseph O'Dwyer, of New York.

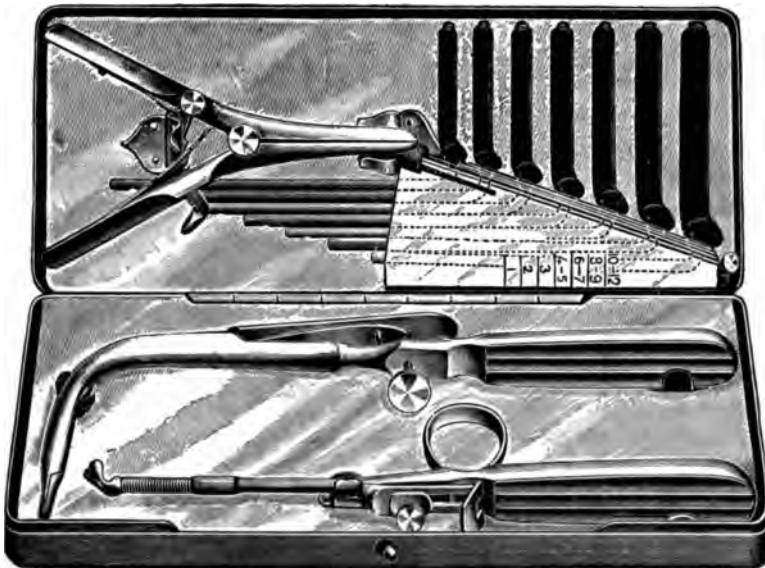


FIG. 199.—O'DWYER'S INTUBATION SET.

A set of O'Dwyer's instruments (Fig. 199) consists of seven tubes, an introducer, an extractor, a mouth-gag, and a gauge. The tubes are made of hard rubber and lined with gold-plated metal. So carefully did

O'Dwyer perfect his instruments that nothing of importance has been added by others. It is interesting to note that nearly all the modifications which have been suggested since his first publication had already been tried by him and discarded. No one thing is more essential to success with intubation than properly constructed instruments. The operation is not difficult, if one has had practice on the cadaver. Without this it should not be attempted. The tube is selected according to the age of the patient, this being indicated on the gauge. A very large child will often require a tube of larger size than its age would call for.

**Introduction of the Tube.**—Either one of two positions may be employed, the choice depending upon the preference of the operator. Formerly the usual method was to have the child seated upon the lap of a nurse while his head was steadied by a second assistant standing behind. In the other position the child lies upon his back upon a table, his head being steadied by an assistant. In both positions the arms should be pinioned to the sides by a sheet. In the recumbent position the child can be held more firmly; it has also the advantage of dispensing with one assistant, and in an emergency with both of them. The tube is attached to the introducer, and the gag is inserted into the left angle of the mouth and opened as widely as possible. The slipping of the gag and laceration of the mouth may be prevented by using a piece of rubber tubing to cover each arm of the gag where it comes in contact with the gum. The attempts at introduction must be made quickly, for during them respiration is practically arrested. Several short attempts are always better than a single prolonged one. Very little force is ordinarily required in introducing the tube, that used in passing a catheter being a good general guide. In cases of subglottic stenosis, however, quite a little force may be necessary.

The index finger of the left hand is used as a guide in introduction. This is passed well back into the pharynx, then brought forward until a hard nodule—the upper border of the cricoid cartilage—is encountered. This is the best of all landmarks, since the soft parts are often distorted by swelling. Directly in front of the cricoid cartilage may be felt the epiglottis and the opening of the larynx, which are readily recognised after the touch has become somewhat educated. The epiglottis is drawn forward and the tube is passed along the palmar surface of the left index finger, by which it is guided into the larynx; it is then pushed off the introducer by a thumb-piece attached to its handle. When it is certain that the tube is in position, and the patient breathes properly, the loop of silk attached to the head of the tube is cut off and pulled through, the removal of the tube being prevented by placing the left forefinger upon its head. The silk is not usually left attached unless there is evidence of loose membrane below the tube. It may be desirable to leave the silk attached in case no one can be within reach who is able to remove

the tube should it become obstructed. The child's arms and hands should then be secured to prevent him from seizing it himself. When not removed the silk is fastened to the cheek by a piece of adhesive plaster. The tube is known to be in place, first, by the hissing breathing sounds, somewhat similar to what is heard when the trachea is opened; secondly, by a severe paroxysm of coughing, which is usually excited by a tube in the larynx; thirdly, by the relief of the dyspnoea. If this relief is not very apparent the physician may still be in doubt as to whether the tube is in the larynx or the œsophagus. If in the former, it can not be pushed down by the finger without depressing the larynx with it; and by introducing the finger into the pharynx, the posterior wall of the larynx can be felt between the finger and the tube. The most common mistake made is to pass the tube into the œsophagus. This sometimes happens because the position of the child's head is improper—too far forward or too far backward—but more often because the operator has not been quite sure of his landmarks. If this has occurred, there is no relief to the dyspnoea, no hissing sound, and the tube can be pushed down indefinitely. When this condition is recognised, the tube is withdrawn by the loop of silk and after a few moments a second attempt made.

False passages in the larynx are most frequently made by employing too much force or because the operator has worked at the angle of the mouth instead of keeping in the median line. The tube usually goes into one of the ventricles, and may be pushed quite through the larynx into the cellular tissue. This is not likely to happen unless undue force has been used. The production of a false passage is recognised by the fact that, although the tip of the tube can be felt to enter the larynx, it does not descend, but projects above the epiglottis.

False membrane which has become loosened is sometimes crowded down by the tube and obstructs the larynx just below it. This is one of the most serious accidents that may occur, but fortunately it is not a frequent one. It is more likely to happen when the disease has existed for several days than in recent cases. The tube may be in place in the larynx as shown by all the signs above mentioned, except relief of the asphyxia. In such a case the immediate withdrawal of the tube is necessary, it being often followed by the discharge of masses of loose membrane. This is aided by the administration of half a teaspoonful of pure whisky or brandy to excite a strong cough. Artificial respiration may be required, and if there is no relief by any of these means tracheotomy is indicated. Asphyxia is sometimes produced by prolonged and injudicious attempts at intubation.

*After-treatment.*—So far as the tube itself is concerned no treatment is required. The original disease is to be treated as before. The operation has removed only one danger from the patient, viz., that of asphyxia from mechanical obstruction of the larynx. A good expulsive



cough should occur after the tube is in place. This is necessary to clear the tube of mucus, as the pharynx and larynx are generally filled with it as a result of the manipulation.

The child should not be allowed to lie upon his face, nor should he be held over the nurse's shoulder face downward, for in either position a slight cough is enough to expel the tube. Nursing infants may sometimes continue at the breast after the operation; ordinarily they have but little difficulty in swallowing. Older children often experience considerable trouble in taking liquids. This may be overcome by the device suggested by Casselberry, of having the patient's head lower than his body while he drinks. When fluids cause excessive coughing or at other times when they can be taken only with the greatest difficulty, they may be given through a nasal tube or one passed through the mouth. Semi-solid articles, such as condensed milk, wine jelly, corn starch, ice cream, or scrambled eggs, may be well taken when fluids are not. Feeding is always easier after the first day or two, and patients who wear a tube for chronic disease soon experience no trouble whatever, showing that the difficulty depends more upon the inability to co-ordinate the movements of the muscles of deglutition when the tube is in place than upon mechanical causes, for the head of the tube is effectually covered by the epiglottis.

It sometimes happens that the tube is coughed out soon after its introduction, because too small a size has been used. In some cases this occurs repeatedly. It happened in a case of my own twenty-eight times during four days. Such cases are probably due to paralysis of the laryngeal muscles. The dyspnoea does not usually return for two or three hours after the tube has been coughed out, but may come back at once. It may happen that the tube is coughed up and not seen by the nurse, or it may be coughed up and swallowed by the child. When called because of dyspnoea after operation, the physician should make a digital examination of the pharynx to be sure that the tube is still in place. Swallowing the tube generally causes no harm to the child, for tubes have repeatedly passed through the intestines.

The entrance of food into the bronchi through the tube is a danger that does not exist, and broncho-pneumonia following intubation does not depend upon the entrance of food into the bronchi.

Deep ulceration at the head of the tube very rarely occurs, provided properly made tubes are employed, but superficial ulceration almost invariably is produced at the base of the epiglottis and in the trachea at the lower end of the tube. Deep ulcers extending to the tracheal rings may occur in ill-conditioned children, usually in connection with other complications serious enough to cause death.

Spontaneous descent of the tube into the larynx is almost impossible, and it can not be crowded down without using considerable force and severely lacerating the larynx.

Sudden blocking of the lower end of the tube by membrane loosened from the trachea or bronchi occasionally occurs. The usual result of this is the immediate expulsion of the tube by coughing, the discharge of the loose membrane following. This condition is one of the safety valves of the operation. One of the strong points in favour of intubation is that the forcible cough which the patient is able to make on account of the narrow opening of the tube, often enables him to expel large accumulations of mucus, and even membrane, more readily than through a much larger tracheal opening.

The period for which the tube is required varies much in different cases. It is the experience of practically all operators that it has been materially shortened by the use of antitoxine. The average time of wearing the tube is about five days, and in many it can be dispensed with in two or three days. Should the tube be coughed out at any time, its introduction should be delayed until dyspnoea returns.

**Removal of the Tube—Extubation.**—This is rather more difficult than its introduction. The general arrangement of the patient and assistants is the same as for introduction. The left index finger is placed upon the head of the tube, which is steadied externally by the thumb of the same hand. The beak of the extractor is introduced within the opening of the tube, its jaws are then separated by pressure upon the lever at the handle, and the instrument withdrawn, very slight force being required.

The tube is first removed tentatively, the physician waiting to see if dyspnoea returns. It is well to give a full dose of morphine an hour before the removal of the tube, since this operation is almost invariably followed by a marked degree of laryngeal spasm which lasts for ten or fifteen minutes. To avoid the production of vomiting and the entrance of food into the larynx, food should not be given for two hours previously. If dyspnoea does not return in the course of three or four hours, the probabilities are that the tube will no longer be required. It is exceptional that the patient has great difficulty in dispensing with the tube, as so often happens after tracheotomy.

The only objection of much force urged against intubation is that asphyxia may be produced by crowding down loose membrane into the larynx. This is an infrequent accident; should it happen, and the asphyxia not be relieved by removing the tube and inserting another, tracheotomy may be performed.

There is always some degree of hoarseness following intubation, but in the majority of cases it disappears within a week, occasionally it continues as long as three or four weeks, but it is very rarely if ever permanent. The duration of the aphonia seems to have little relation to the length of time the tube is worn unless this is many weeks.

Experience has clearly proved that intubation relieves the dyspnoea due to laryngeal stenosis promptly, efficiently, and certainly; it does this

without many of the dangers and objectionable features of tracheotomy, while at the same time it does not deprive the patient of any essential advantage which tracheotomy affords.

**Retained Intubation Tubes—Prolonged Intubation.**—Difficulty is experienced in dispensing with the intubation tube much less frequently than with the cannula after tracheotomy; yet when this condition occurs it is the cause of much concern and even danger. Trouble of this sort is seen in about one per cent of the cases of intubation. In the majority of these the patient is able to do without the tube in a few weeks, and such cases require very close attention, but no special treatment other than the substitution at times of a special O'Dwyer tube with an extra large "retaining swell." But occasionally there are met with cases in which every effort to dispense with the tube proves futile. Although the children breathe well with the tube in place, still if it is removed or expelled by coughing, in a short time, varying from a few minutes to several days, the dyspnoea returns with such severity that the tube must be replaced to prevent asphyxia. Inasmuch as these patients sometimes expel the tube several times a day, surgeons have often resorted to tracheotomy to avert the danger of suffocation, which might easily occur if no one were at hand who could replace the tube. This operation, however, gives only temporary relief. Many of these children, after wearing tubes of one sort or another for years, ultimately die from some accident connected with the tube or from pneumonia.

The causes and the exact pathological condition underlying this difficulty are subjects regarding which there has been much difference of opinion. The cause of the returning dyspnoea is probably subglottic swelling and œdema which occur in tissues which are the seat of chronic inflammation as soon as the pressure of the tube is removed. In a few cases a cicatricial condition, the result of previous ulceration, has been found; but it is doubtful if granulations, so frequent a cause of retained cannula after tracheotomy, play a part. The chronic inflammation of the mucous and submucous tissues of the subglottic region of the larynx which produces the symptoms, is aggravated by a faulty tube or a clumsy operation, but it may occur under the most favourable conditions.

For the relief of this condition, O'Dwyer advised in recent cases the application of astringents by means of an intubation tube coated with gelatine with which some astringent was combined. For those patients who cough out the tube frequently, tracheotomy is at times a necessity to prevent sudden death. But this does not affect the original condition, for the same difficulty exists in doing without the tracheal cannula. The operations of laryngotomy, curetting, etc., have been such signal failures as to discourage one from repeating them.

The most successful method of treatment thus far proposed is that of Rogers, which consists in increasing intra-laryngeal pressure by the

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insertion of larger and larger intubation tubes. This is not to be adopted until long after all acute symptoms have subsided. The first tube used is as large a one as can be introduced without force; after a few weeks, the next larger size, and after a longer interval, possibly a still larger one. When the very large tube has been worn for several weeks one is usually able to dispense with all tubes.

True cicatricial stenosis may best be relieved by opening the trachea and dilating from below, and afterward inserting an intubation tube. When there is complete destruction of the cricoid cartilage, as sometimes occurs, tracheotomy is the only remedy, but this is only palliative, as the tube must be worn permanently.

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## CHAPTER IX.

### TYPHOID FEVER.

TYPHOID FEVER is an acute infectious disease due to a specific germ—Eberth's bacillus. It may affect the fœtus *in utero*, or the newly-born child, and it is seen in infancy and throughout childhood.

*Fœtal Typhoid.*—Morse has collected the evidence bearing upon fœtal infection, from which the following conclusions seem warranted: Infection of the child from the mother is a frequent but not an invariable occurrence. The bacilli may pass directly from the maternal into the fœtal circulation. The fœtal form of the disease is a general blood-infection, since the intestines are not functionally active. The most common result is death of the fœtus and consequent abortion; but the child may be born alive still suffering from infection, and die in a short time because of its feeble resistance.

*Infantile Typhoid.*—Much difference of opinion exists regarding the frequency with which typhoid fever occurs in infancy. Some clinicians hold the opinion that the disease is of very common occurrence, but is often unrecognised because of the absence of many of the symptoms which are characteristic at a later age. They regard every protracted fever not malarial and not dependent upon a local inflammation as presumably typhoid. The symptoms from which we may regard the question of typhoid as established will be considered under Diagnosis. I have seen but two undoubted cases of typhoid under two years of age, and I believe it to be rare, at least in New York. No case recognised as typhoid occurred in a child under two years of age during my eight years' service in the New York Infant Asylum, where about 10,000 cases of acute illness were treated and over 700 autopsies made. No case has been recognised as typhoid, either in the wards or the post-mortem room of the New York Foundling Hospital during the past twenty-five years. Typhoid

has been seen by Murchison at six months and by Ogle at four and a half months, the diagnosis being in both instances confirmed by autopsy; also by Griffith at seven months and by Taylor at eight months, with fairly typical symptoms. It is during epidemics that most of the infantile cases are seen; sporadic instances of infantile typhoid should always be regarded with suspicion, and I believe that most cases so diagnosticated are questionable. Even in epidemics it is surprising that so few infants are attacked. In that of Montclair, N. J., in 1894, of 115 cases, only 3 were under two years; in that of Stamford, Conn., in 1895, of 406 cases only 4 were under two years.

*Typhoid in childhood* is by no means rare, but it is not until after the fifth year that it can be said to occur frequently. The following figures, embracing groups of cases reported by eight writers, represent the relative frequency with which the disease is seen at the different ages: Of 970 cases, eight per cent occurred under five years, forty-two per cent between five and ten years, and fifty per cent between ten and fifteen years.

Typhoid fever is almost invariably contracted by drinking water or milk which contains the germs of the disease. The infrequency of typhoid in infants is explained, in part at least, by the fact that most of the water and a large part of the milk taken are previously boiled, or heated in some manner.

**Lesions.**—Typhoid in young children is so seldom fatal that opportunities for a study of the lesions have been limited. In a general way they resemble those of adults except in severity. In a considerable number of the cases the pathological process in the intestines does not go on to ulceration; and when ulcers form they are seldom large or deep, and perforation is very rare. Montmollin gives the following facts concerning twenty-three autopsies, most of them, however, being in children over eight years old: ulcers were present in seventeen cases; they were situated in the lower ileum in sixteen, and in ten they were only there; in the ascending colon in nine, and only there in one case; perforation occurred in three cases, in every instance in the lower ileum. Autopsies made upon infants may show even less severe intestinal lesions than those mentioned. In fact, some cases in which the clinical diagnosis was beyond question, have shown only moderate redness and swelling of Peyer's patches, the solitary follicles and the mesenteric lymph nodes—lesions which are exceedingly frequent in cases of simple diarrhoea. In a doubtful case such post-mortem findings do not establish the diagnosis of typhoid. Indeed, they prove nothing unless cultures from the intestinal contents, the mesenteric glands, or other organs, show the typhoid bacillus. Enlargement of the spleen is practically constant. The degenerative changes in the heart, the kidneys, and the liver are much less frequent and generally less severe than in adults.

**Symptoms.**—The peculiar features of typhoid in early life are seen only in children under ten years old; for after this time the disease does not differ essentially from the adult type. In brief, the typhoid of early childhood may be described as a fever characterised more often by nervous symptoms than by intestinal symptoms.

**Onset.**—A sudden onset with well-marked symptoms—fever, prostration, vomiting, etc.—is not uncommon; in fact, it is quite as frequently seen as the insidious beginning, with lassitude, headache, coated tongue, anorexia, and gradual rise in temperature. In cases developing abruptly it often appears as if an acute indigestion had been the means of precipitating the attack. The most frequent initial symptom is vomiting; a chill is rare. Epistaxis occurs as an early symptom rather less frequently than in adults.

**Condition of the Bowels.**—There is no constant relation between the severity of the intestinal lesions and the condition of the bowels. Taking large groups of cases together, diarrhoea is present in about half the total number. It is rarely profuse, from two to four discharges a day being the average. The appearance of the stools is seldom characteristic; they are usually thin and fluid, often containing mucus. Constipation may be present at the beginning only, or throughout the attack. Tympanites is generally moderate, and is often entirely absent; it usually accompanies constipation. Marked iliac tenderness and gurgling are infrequent.

**Spleen.**—By the end of the first week this is almost invariably found to be enlarged to a sufficient degree to be recognised by palpation. Usually the spleen extends but an inch or an inch and a half below the ribs, but at times it may be three inches or more; persistent enlargement always indicates that the disease is not at an end even though the temperature has reached the normal, and a relapse should be expected.

**Eruption.**—It is the experience of nearly all who have seen much of typhoid in children that the eruption is less constant, less abundant, and less characteristic than in adults. The typical eruption consists of small, scattered, rose-coloured spots, which appear chiefly or solely upon the abdomen at the beginning of the second week. They come in successive crops, each one of which generally lasts three days, the whole duration of the eruption being about ten days.

**Prostration, Emaciation, etc.**—As a rule the prostration is quite sufficient to keep a child in bed after the first few days. The general weakness after this time is in direct proportion to the height of the temperature. Loss of flesh is steady and usually marked; and in a prolonged attack there may be extreme emaciation.

**Temperature.**—In the cases with a gradual onset, the typical temperature curve is one which rises steadily for from two to seven days, fluctuates within the limits of one to three degrees during the second

week, and steadily declines during the third week, reaching the normal on the average at the end of the third week. In cases with an abrupt onset, the temperature rises at once to from 102.5° to 105° F., but subsequently may run the same course as in the first group.

The following are the most important variations from the temperature curve of adults: The initial rise is much more frequently rapid;

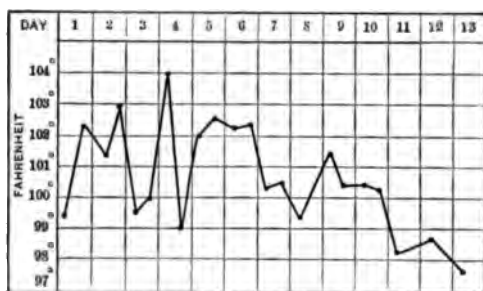


FIG. 200.—TYPHOID FEVER OF SHORT DURATION IN A CHILD THIRTEEN MONTHS OLD. Spleen enlarged; eruption typical; no diarrhoea and only moderate abdominal distention. There were two other cases in the family, all being due to the same cause—infected milk. (After Northrup.)

during the second week the remittent character is less marked, this probably depending upon the fact that ulceration is less frequent and less extensive; the average duration is shorter. In young children the proportion of cases in which the fever lasts only from eight to fourteen days is quite large (Fig. 200). After the age of ten years the type of the fever is much like that seen in adults.

The maximum temperature in the mild cases is 103° or 104° F.; in the severe ones it often reaches 105° or 106° F., but rarely goes above this point. The range is usually higher than in adult cases of the same severity. At the beginning of convalescence a subnormal temperature is very frequent, and by many writers is considered to be the rule. A secondary rise is most frequently due to errors in diet, but may occur from the development of complications. A sudden fall indicates either perforation or intestinal hæmorrhage.

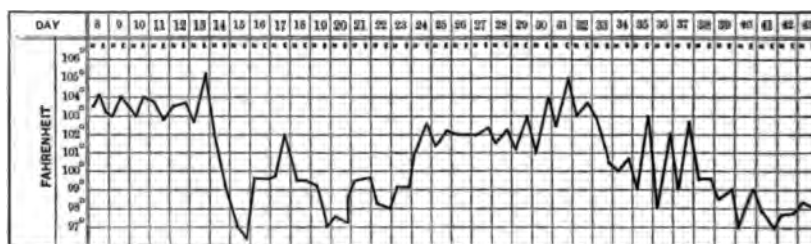


FIG. 201.—TYPHOID FEVER WITH RELAPSE. Child two and a half years old; early temperature high and symptoms typical; natural fall on fourteenth day; rise on seventeenth day apparently due to otitis; relapse on twenty-fourth day, with fresh eruption and return of splenic swelling which had disappeared. Temperature was subnormal at the end both of primary and secondary fever.

*Relapses* were present in 8.4 per cent of 533 cases collected by Morse. They follow about the same course as in adults (Fig. 201).

*Nervous Symptoms.*—As a rule, these are more prominent in severe cases than the intestinal symptoms, and are directly proportionate to the height of the temperature. The extreme nervous symptoms belonging to the typhoid state in adults are rare in childhood, except in patients over ten years old. Headache and mild delirium at night are very frequent, the former being seen in the majority of cases. Young children are usually dull, apathetic, and often in a state of semi-stupor. Occasionally the disease may closely simulate meningitis. The nervous symptoms are usually most severe in the second, or early in the third week, and subside as the temperature declines.

*Pulse.*—This is increased in frequency, but not to the degree that is seen in most diseases of childhood with a similar elevation of temperature. The force and rhythm of the pulse are usually good, irregularity and dicrotism being rare in children as compared with adults.

*Urine.*—A small amount of albumin is found in the urine of most of the severe cases at the height of the disease, and is due to acute renal degeneration; but a marked degree of nephritis is infrequent. In from one-fourth to one-third of the cases typhoid bacilli are found in the urine, generally in pure culture. They usually appear in the latter part of the disease, the second or third week, and may continue for months or even years. They are sometimes accompanied by evidence of cystitis or nephritis. Their number is in some cases so large as to render the urine turbid; in others they give no indication of their presence. Ehrlich's diazo reaction is usually present at the height of the fever.

*Blood.*—The characteristic blood picture in typhoid is a low leucocyte count, generally under 10,000, accompanied usually by a slightly increased proportion of lymphocytes. Blood cultures, with great uniformity, show the bacilli even in the first week of the disease.

*Intestinal Hæmorrhage.*—Of 946 collected cases, mainly from hospital reports, intestinal hæmorrhage occurred in thirty, or about three per cent; the majority of these were in children over ten years old. Of twenty-four collected cases of hæmorrhage in children, ten terminated fatally. The youngest case of this nature which has come under my own notice was in a child of four and a half years.

*Intestinal Perforation.*—This is even more rare than hæmorrhage. In 1,028 collected cases, this accident occurred but twelve times, or in 1.1 per cent. Perforation is indicated by a sudden fall in the temperature, with collapse; usually there is vomiting and the rapid development of tympanites.

*Complications and Sequelæ.*—The complications of typhoid in early life are infrequent and usually mild. Bronchitis is present in most of the severe cases. Pneumonia has been noted in nine per cent of the cases reported by various authors. Both serous and purulent effusions into the chest are occasionally seen, and sometimes abscess of the lung.



Complications referable to the nervous system are not very frequent, but are of much interest. Meningitis is extremely rare. Morse has collected twenty-one cases of aphasia, in two of which it was clearly due to embolism; in the remainder, however, it apparently was not dependent upon any organic lesion. In two-thirds of the cases it came on during convalescence, and in nearly all complete recovery occurred after an average duration of three weeks. Aphasia usually followed a severe type of the disease, and in most of the cases was not accompanied by any other paralysis or by mental disturbance. Insanity is a rare sequel of typhoid in children, the usual type being acute mania. Adams (Washington) has reported two examples of this, both terminating in recovery. Chorea is seen rather oftener than after the other infectious diseases.

Otitis is not an infrequent complication, occurring much oftener than in adults. It is principally seen in young children and during the cold season. Among the less frequent complications may be mentioned: parotitis, which is usually suppurative and is seen in septic cases; abscess of the liver, examples of which have been reported by Bokai, Asch, and others; gangrenous inflammation of the mouth or genitals; pericarditis, endocarditis, and peritonitis, suppurative inflammations of joints, multiple abscesses and furunculosis. Tuberculosis of the lungs or bones not infrequently follows typhoid.

**Diagnosis.**—The diagnostic symptoms of typhoid are the Widal blood reaction, the discovery of the bacilli in the blood, urine or fæces, the eruption, the course of the temperature, the enlargement of the spleen and the abdominal symptoms—diarrhœa, tympanites, intestinal hæmorrhage, and perforation.

The Widal reaction is present at some period in from ninety-five to ninety-eight per cent of the cases, and thus becomes the most valuable single symptom for diagnosis. It is seldom obtained before the seventh day and frequently not until the tenth; it may not be present until convalescence or a relapse. Repeated tests should always be made if the first reaction is negative or doubtful; and the tests should be made by an experienced pathologist. The reaction is therefore of much less value for an early than for an exact diagnosis. A positive reaction may be present if the patient has previously had typhoid, something much less likely to be the case with children than with adults; in rare instances it has been obtained in other diseases or in health when no history of previous typhoid existed. Both these conditions, however, are very exceptional, and a positive reaction may as a rule be taken to establish the diagnosis.

Typhoid bacilli, according to the observations of Park, may be demonstrated in the stools by culture in a large proportion of the cases. They are found in the urine, usually in the latter part of the disease, in about one-third the cases. Their discovery in either of these discharges

is conclusive evidence of previous or existing typhoid. An examination of both urine and *faeces* should, if possible, be made in all doubtful cases.

The course of the temperature is an important aid to diagnosis, but alone is not to be depended upon. The characteristic feature is a fever which continues for two, three, or four weeks, and subsides spontaneously. The variations from the adult type have already been mentioned, also the frequency of the eruption, the enlargement of the spleen, and the abdominal symptoms. We are not warranted, I think, in making the diagnosis of typhoid, if repeated tests fail to show the Widal reaction or if the eruption and splenic enlargement are absent, and no bacilli can be demonstrated in the discharges, no matter what the course of the temperature may be.

One should hesitate to make the diagnosis of typhoid in a child under two years old, unless the disease is epidemic. The great majority of sporadic cases reported as occurring in infancy are probably not typhoid. After the fifth year the disease is more frequent, and its symptoms in general resemble those of adults, except in severity.

A differential diagnosis is to be made from malarial fever, ileo-colitis, meningitis, tuberculosis, and from other ill-defined continuous fevers of unknown origin. From malarial fever the diagnosis is to be made by the temperature curve, the organisms in the blood, and the effect of quinine. In most of the cases of malaria the temperature will be found to touch the normal at some time in the twenty-four hours. The administration of full doses of quinine is a diagnostic test of much practical importance; an irregular or remittent fever which yields promptly to quinine is most certainly not typhoid.

Ileo-colitis and typhoid fever are not often confounded. The former is chiefly seen in the first three years of life, a time when typhoid is rare. The intestinal symptoms of ileo-colitis are marked even though the temperature is not high, and they are altogether more severe than is usual in typhoid; while enlargement of the spleen, tympanites, and the eruption are not present.

The cerebral symptoms of typhoid may be difficult to distinguish from meningitis, unless one has watched their development. Irregular respiration, a slow, irregular pulse, localised paralysis and complete coma are seldom, if ever, seen in typhoid, and a retracted abdomen very rarely, while the enlarged spleen and the peculiar eruption are not seen in meningitis. In typhoid with pronounced nervous symptoms the temperature is usually higher than in meningitis.

General tuberculosis very often resembles typhoid so closely that a differential diagnosis is almost impossible until local signs of tuberculosis have appeared, usually in the lungs.

**Prognosis.**—Of 2,623 cases in children, collected from the reports of twelve different writers, the mortality was 5.4 per cent. These are, how-

ever, almost all taken from hospital reports, where as a rule the mildest cases are not brought for treatment. The mortality of the disease in children over three years old probably does not exceed three or four per cent. Death seldom occurs from the disease itself, but usually from some accident or complication, the most frequent being pneumonia and intestinal hæmorrhage or perforation. Griffith's collection of cases occurring in infancy indicates a much higher mortality for this period. The death-rate for the first year reached nearly fifty per cent.

**Treatment.**—The usually low mortality of this disease shows how successful all methods of treatment are likely to be considered. In the great majority of cases very little active treatment is required. Every patient with typhoid should be put to bed and kept there during the febrile period, and a few days beyond it, no matter how mild the attack may be. The diet should consist of sterilised milk or animal broths, cereal gruels and very soft eggs. These articles should be given regularly every three hours, but not pushed beyond the desire of the patient. Milk may be diluted or partially peptonised, and kumyss or matzoon may be substituted for it if the stomach is irritable. Plenty of water should be given. Solid food should not be allowed until the temperature is normal.

Both the urine and faeces should be immediately and thoroughly disinfected by a solution of carbolic 1:20. If the movements are in a chamber or a bed-pan they should be covered with this solution for at least six hours before they are thrown into the water-closet. If napkins or diapers are used, they should be soaked in some effective antiseptic solution for twelve hours and then thoroughly boiled. Sheets stained by discharges should be treated in the same way, and all bed-linen should be boiled for two hours apart from the washing of the family. The efficiency of hexamethylenamine (urotropin) in removing typhoid bacilli from the urine seems now to be well established. It should be given at the close of the attack in doses of three to five grains, three times a day, and continued for a week or ten days.

Diarrhœa calls for treatment only when the movements exceed four or five in twenty-four hours. If no more than this number are present, they should not be interfered with. Opium and bismuth are undoubtedly the best means for controlling excessive diarrhœa, but care should be taken that they are not pushed to the degree of inducing constipation.

Constipation early in the disease may be relieved by calomel, followed by the salines, or castor oil, but all active purgation should be avoided. Later in the disease daily irrigation of the colon with tepid water is better than anything else. On the whole, constipation is more troublesome to overcome than diarrhœa.

Tympanites is rarely severe enough to require treatment; it may be relieved by turpentine stupes, by a glycerin suppository, or a small

glycerin injection (one teaspoonful of glycerin to two ounces of water), or, better still, by the use of the rectal tube.

Whenever the temperature remains above 104° F., antipyretic measures are indicated. In mild cases cold or tepid sponging is generally sufficient. In those which do not yield to such measures, baths may be employed. Not all children bear baths well, and considerable discretion should be used in employing them. One should be guided quite as much by the effect upon the pulse and the nervous system as by the temperature. The best method is usually the graduated bath; the child is placed in the tub with the water at a temperature of 95° or 100° F.; this is gradually lowered to 95°, 90°, or even 85° F., but seldom lower. The body should be actively rubbed while the child is in the bath, to prevent shock and cardiac depression. The cold pack may be substituted for the bath where circumstances make the latter impracticable. The bath or pack should be repeated in an average case in from three to six hours.

The milder nervous symptoms—headache, restlessness, sleeplessness, etc.—may be relieved by an occasional dose of phenacetine, either alone or in combination with the bromides, or by cold or tepid sponging; the more severe ones usually occur with high temperature, and are best controlled by the cold bath.

Stimulants in most of the cases are not called for. They are to be given according to the indications afforded by the pulse, the first sound of the heart, and the child's general condition. They are seldom needed earlier than the end of the second week. Intestinal hæmorrhage calls for absolute quiet, morphine hypodermically, and an ice-coil to the abdomen, nothing being given by mouth except stimulants, turpentine, and possibly opium. Intestinal perforation is successfully treated only by early laparotomy.

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## CHAPTER X.

### TUBERCULOSIS.

TUBERCULOSIS is an infectious communicable disease, due to the bacillus tuberculosis of Koch. It may be local or general, and may involve any organ and almost any structure in the body.

**Etiology.**—*Age and Frequency.*—No age is exempt from tuberculosis. It was formerly believed that the disease was rare in infancy, but recent observations have shown the opposite to be the case.

Statistics taken chiefly from three New York institutions where only infants and young children are received give the following figures for 382 cases of tuberculosis, the diagnosis being confirmed by autopsy in nearly every instance: In the first year there were 160 cases, and of these 67 were under six months, 10 of which were under three months

of age. The frequency of tuberculosis appears to increase steadily as age advances, as shown by the following table, in which results at autopsy are compared with those obtained by means of v. Pirquet reaction. Both series are from Vienna.

| Age.                   | I. Hamburger: Autopsies. |                             | II. v. Pirquet: Skin |                   |
|------------------------|--------------------------|-----------------------------|----------------------|-------------------|
|                        | No. of Cases.            | Percentage of Tuberculosis. | No. of Tests.        | Percent Reaction. |
| Under 3 months.....    | 105                      | 4 per cent                  | 147                  | 0 per cent        |
| 3 to 6 ".....          | 73                       | 18 "                        | 64                   | 5 "               |
| 6 to 12 ".....         | 140                      | 23 "                        | 67                   | 16 "              |
| 2d year.....           | 179                      | 40 "                        | 88                   | 24 "              |
| 3d and 4th years.....  | 175                      | 60 "                        | 127                  | 37 "              |
| 5th and 6th years..... | 67                       | 56 "                        | 101                  | 53 "              |
| 7 to 10 years.....     | 65                       | 63 "                        | 182                  | 57 "              |
| 11 to 14 ".....        | 44                       | 70 "                        | 100                  | 68 "              |
| Over 14 ".....         | ...                      | ...                         | 112                  | 90 "              |
|                        | 848                      | 40 per cent                 | 988                  | 41 per cent       |

From the facts at hand it would seem that the percentage of children with tuberculosis is much greater in Europe than in this country. The following table gives figures for three institutions with which I am connected in New York, as compared with data taken from Vienna and Munich.

*Frequency of Tuberculosis as Shown by Autopsies.*

| INSTITUTION.               | AGE OF PATIENTS.                                | No. of Autopsies. | No. showing Tuberculosis. | Percent Tuberculosis. |
|----------------------------|---|-------------------|---------------------------|-----------------------|
| N. Y. Infant Asylum..      | Nearly all under 2½ years.                      | 726               | 56                        | 8.0 per cent          |
| Babies' Hospital.....      | " " " 3 "                                       | 1,000             | 168                       | 16.8 "                |
| N. Y. Foundling Hosp. .... | " " " 3 "                                       | 1,000             | 136                       | 13.6 "                |
| Müller—Munich.....         | Children of all ages.....                       | 500               | 200                       | 40.0 "                |
| Hamburger—Vienna.....      | All ages up to 14 years.....                    | 848               | 335                       | 40.0 "                |
| " " ".....                 | (Including only children of 2 years and under.) | 497               | 120                       | 24.4 "                |

These percentages are not to be taken to represent the occurrence of tuberculosis in the community generally, but only its frequency in the class which furnishes hospital and institution inmates. Nor are these figures to be interpreted as showing the percentage of active tuberculosis. In the cases showing tuberculosis at autopsy nearly one-third of the number died from other diseases, tuberculosis being latent, its existence being discovered only post mortem. Likewise in not more than one-fifth the cases giving positive skin reactions there were no evidences of active tuberculosis.

*Predisposing Causes.*—These include all the conditions which tend to about a diminished resistance of the body to tuberculous infection.

susceptibility may be inherited, as when parents have suffered from tuberculosis or other constitutional disease, syphilis, alcoholism, etc. It may be due to the fact that children have been reared in crowded city tenements, in institutions, or under other unfavourable surroundings. A local predisposition may be afforded by any pathological condition of the organs or mucous membranes exposed to infection. Thus, adenoid growths of the pharynx or large tonsils may favour the development of cervical adenitis, and frequent attacks of bronchitis may precede pulmonary tuberculosis. Certain infectious diseases, particularly measles, whooping-cough, and influenza, greatly increase a child's susceptibility to tuberculosis, and these may also cause a latent tuberculosis to develop into an active process. General or pulmonary tuberculosis is therefore often seen as a sequel to the diseases mentioned, the latent focus for which has been tuberculous bronchial glands.

*Modes of Infection.*—Intra-uterine infection, although rare, has been established by the report of at least seven complete and well-authenticated cases. Tuberculosis of the placenta is more frequent. In most of the cases of congenital tuberculosis the mother has been suffering from the disease in an advanced form, and the child is either still-born or dies soon after birth. Besides tuberculosis of the placenta, tubercle bacilli are found in the organs of the child, and, when life is prolonged, there are generalised lesions showing infection through the blood. Cheesy nodules have been observed in the umbilical cord. Intra-uterine infection is highly probable in many of the children born of tuberculous mothers, who develop the disease during the first few months of life, although they may show no evidence of it at birth. Among my own cases there was one only twenty days old. The child was born prematurely of a mother suffering from advanced tuberculosis. Besides other lesions, the autopsy showed, in the case of the mother, tuberculosis of the endometrium.

Tuberculosis may be communicated by direct inoculation, as in the case of a bite from a person suffering from the disease, several instances of which are on record. The rite of circumcision performed by a rabbi suffering from tuberculosis is also known to have caused the disease. One of the most striking instances of direct infection is that reported by Reich.<sup>1</sup> In a town of about 1,300 inhabitants, the obstetric practice was divided between two midwives. Within fourteen months no less than ten infants, who had been delivered by one of these women, died of tuberculous meningitis. In none of these families was there a history of tuberculosis. This midwife was found to be suffering from pulmonary tuberculosis, and died from that disease. It was her custom to remove the mucus from the mouth of the newly-born infants by direct mouth-

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<sup>1</sup> Berliner klinische Wochenschrift, No. 37, 1878

to-mouth aspiration, and then to establish respiration by blowing the nose. In the practice of the other midwife, who was healthy, cases of tuberculosis occurred, although she treated the newly-born infants in the same fashion.

I believe that altogether the most frequent means by which children acquire tuberculosis is from association with persons suffering from pulmonary tuberculosis. Some of these are persons in the active stage of the disease; many are supposed to have been cured; in others the disease has not yet developed so as to be recognised. Bacilli may be directly conveyed by kissing. Dried sputum containing bacilli may become part of the dust of the room; it may be inhaled or it may be introduced into the mouths of children by hands, toys, or other objects. The source of infection is usually one or other parent or some member of the household—a nurse, caretaker, servant, or a frequent visitor. A history of such exposure was definitely traced in forty-four per cent of 101 consecutive cases of tuberculosis in young children which were investigated at the Babies' Hospital. These figures do not represent the proportion of the cases in which the disease is so contracted. I believe there is a very much larger number in which this connection can not be traced. Doubtless exposure antedates symptoms by a number of weeks, and often by several months. In instances where it could be pretty accurately ascertained, the interval between exposure and development of symptoms was from four to twelve weeks.

Infection may take place from beds, rooms, sleeping cars, or apartments previously occupied by tuberculous patients; from dishes, spoons, from glasses at public drinking places; also from the milk of tuberculous cows<sup>1</sup> or from meat. My own observations lead me to the conclusion that only a very small proportion of children contract tuberculosis in these indirect ways. Infection through milk I believe to be relatively rare. (See chapter upon Milk.) Unless the disease in the animal is far advanced or the udder is involved, the number of bacilli

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<sup>1</sup> In this connection the following incident is interesting as bearing upon the other side of the question: Near a large American city was a fancy stock farm of registered Jersey cows, which supplied milk for table use and infant feeding to a large number of families in the wealthiest part of the city, for a period of over ten years. At the end of that time the tuberculin test was used for the first time, and 45 per cent of these cows were found to be tuberculous, and were killed by order of the State Board of Health. The diagnosis was confirmed by autopsies upon the animals in several instances. An investigation was instituted among the children who had been supplied with this milk, but in only one case of many hundreds could it be learned that tuberculosis had developed, and in this instance it was by no means established that the milk had been the source of infection. It should be stated that this was before the days of sterilising milk for infant feeding. Besides the families who took the milk in the manner mentioned, the employees at the farm were accustomed to drink skimmed milk in large quantities daily as a beverage in the place of water. Many of them continued to do this for years, and yet not one of them developed tuberculosis.

present in the milk of a tuberculous cow is small and the chances of infecting a child are slight. Those which enter may be destroyed in the stomach or pass through the intestinal tract without doing harm. Bacilli entering through the respiratory tract unfortunately have no such ready means of exit. Infection from the meat of tuberculous animals is a possibility, but hardly more. Bollinger's experiments in feeding animals with the expressed juice of such meat gave negative results.

*Types of Bacilli.*—Important information in regard to the source of infection is obtained from a study of the type of organism present in the different varieties of tuberculosis. Of 137 cases of tuberculosis in children investigated by Park and Krumwiede in the Research Laboratory of the New York Health Department the following results were obtained: The human type was found in 107 cases; of which 13 were pulmonary; 29, glandular; 2, abdominal; 33, meningeal; 16 in bones and joints; 1, genito-urinary; and 13 were generalised. The bovine type was found in 30 cases; of which none were pulmonary; 20, glandular; 5, abdominal; 1, meningeal; none, bones and joints alone; 4, generalised.

**Paths of Infection of the Tubercle Bacillus.**—Tubercle bacilli may gain entrance to the body through the respiratory or the alimentary tract or the skin, the last, however, being so rare that it needs only to be mentioned. In infancy and early childhood, infection I believe to be most frequent through the respiratory tract. The situation of the primary lesions strongly supports this view. Bacilli taken in with the inspired air may lodge upon the adenoid tissue of the naso-pharynx and enter the body through the blood or the lymph stream. Such infection is favoured by pathological conditions of these structures. Bacilli which pass the upper respiratory tract may not be arrested until the smaller bronchi are reached. Both clinical experience and animal experiments indicate that the bacilli may pass through a mucous membrane without inducing in it a tuberculous disease, but that penetration is much easier if the mucous membrane is the seat of a catarrhal inflammation, or if the epithelium has been injured. The bacilli are usually taken up by the lymphatics from the surface of the mucous membrane upon which they have lodged, and are carried to the nearest lymph nodes, where they may excite a tuberculous inflammation, but where they may be permanently arrested. The great majority of children who suffer from tuberculosis of the cervical lymph nodes escape general tuberculous infection.

In autopsies both upon children and adults dying from various non-tuberculous diseases it is not infrequent to find tuberculosis limited to the bronchial lymph nodes.

Arriving at the lymph node, the bacilli light up a tuberculous inflammation of varying degrees of intensity, depending upon their number and upon local conditions. This inflammation may pass through the



usual changes of tuberculous glands—congestion, swelling, cell proliferation, and caseation; or the process may be arrested at any point, and the products of inflammation become encapsulated by a proliferated fibrous tissue, in which condition they may remain latent in the glands for an indefinite number of years—possibly for a lifetime. This is often the case in many children, and is consistent with every outward sign of health, but it is a smouldering ember which at any time may be fanned into flame under the stimulus of an inflammation excited by some other cause.

In infants and young children there is a strong tendency for the bacilli to lodge first in the bronchial lymph nodes, probably on account of the favourable conditions for entrance existing in the bronchial glands and lungs. In those who are delicate and have but little resistance, the process in the lymph nodes is likely to go on to caseation and softening, and, secondary to this process in the glands, the lung may become infected. The manner of the extension of the disease to the lung is usually always easy to trace; but in many instances it has been shown to result from the softening of one of these small tuberculous lymph nodes, which then ulcerates through the wall of one of the small bronchial blood-vessel, in this way distributing its bacilli through the lung.

Although this is the course usually taken by bacilli when they are inhaled, it is not always the case. Lesions in the lungs are occasionally found where the lymph nodes are not involved; and there are other cases in which advanced changes exist in the lung, while only the earliest changes are seen in the lymph nodes. In these cases, which perhaps are considered as exceptional, the tuberculous process probably begins in the walls of the small bronchi, the alveoli, or in the connective-tissue septa.

For bacilli which may find their way into the mouth the tonsil may be a portal of entry. Those which pass to the stomach rarely cause lesions of the gastric mucous membrane, or through it reach the systemic circulation. In the intestines, however, more favourable conditions exist. It is possible for the bacilli to reach the mesenteric lymph nodes without causing a lesion of the intestinal mucous membrane. Experiments upon animals have shown that from the intestine they can even reach the bronchial lymph nodes; but in the human subject this is believed both to be exceedingly rare. By careful search I have so far failed to find intestinal ulceration when the mesenteric lymph nodes were manifestly tuberculous.

**Lesions.**—In the following table are given the lesions found in autopsies, of which I have notes. These represent the lesions of infancy and early childhood, seventy per cent of these children being two years old or under. For comparison there are given statistics of 131 autopsies from the Pendlebury Hospital, Manchester, England. Few of the

dren in this series were under three years old. The greater frequency of abdominal tuberculosis, especially tuberculous peritonitis, will be noted. This difference obtains in nearly all the English statistics of the disease.

*Frequency of the Different Visceral Lesions of Tuberculosis.*

| ORGANS.                          | Personal cases;<br>255 autopsies (chiefly under<br>three years). |               | Pendlebury Hospital Reports;<br>131 autopsies (chiefly over<br>three years). |               |
|----------------------------------|--|---------------|--|---------------|
|                                  |  |               |  |               |
| Lungs . . . . .                  | 235  | 92.1 per cent | 122  | 93.0 per cent |
| Pleura . . . . .                 | 93   | 36.5 "        | 100  | 76.0 "        |
| Bronchial lymph nodes . . . . .  | 208  | 81.5 "        | 91   | 70.0 "        |
| Brain . . . . .                  | 85   | 33.3 "        | 60   | 46.0 "        |
| Liver . . . . .                  | 178  | 69.8 "        | 86   | 65.0 "        |
| Spleen . . . . .                 | 191  | 74.9 "        | 76   | 58.0 "        |
| Kidneys . . . . .                | 88   | 30.6 "        | 54   | 41.0 "        |
| Stomach . . . . .                | 7  | 2.7 "         | 1  | 0.8 "         |
| Intestines . . . . .             | 110  | 43.1 "        | 65   | 50.0 "        |
| Mesenteric lymph nodes . . . . . | 118  | 52.4 "        | 77   | 59.0 "        |
| Peritonæum . . . . .             | 22   | 8.6 "         | 37   | 28.0 "        |
| Pericardium . . . . .            | 10   | 3.9 "         | 4  | 3.0 "         |
| Endocardium . . . . .            | 1  | 0.4 "         | ...  | ...           |
| Thymus . . . . .                 | 5  | 1.9 "         | ...  | ...           |
| Suprarenal capsules . . . . .    | 4  | 1.5 "         | 2  | 1.6 per cent  |
| Pancreas . . . . .               | 4  | 1.5 "         | ...  | ...           |

*The Varieties of Tuberculosis seen at Different Ages.*—During the first two years of life, tuberculosis most frequently involves the lungs and bronchial lymph nodes. It is the meningeal or pulmonary process which most often is the cause of death. Death from other forms of tuberculosis is rare at this time of life. Of 232 deaths from tuberculosis in the first three years of life, meningitis was the cause in 93, tuberculous peritonitis in only one, and hæmorrhage from a tuberculous ulcer of the intestine in one.

After the second year, tuberculosis of the bones, cervical and mesenteric lymph nodes, peritonæum, and intestines becomes more frequent, and may occur as the principal lesion, although at autopsy the lungs are usually involved to some degree.

*Pulmonary Lesions.*—As compared with that of adults, the pulmonary tuberculosis of young children is more widely diffused, and the predominance of cases in which the lesion is in the upper lobes is less marked, though it still exists. In those who have passed the sixth or seventh year, the pathological processes resemble those of adult life. Although localised tuberculous processes are frequently met with in patients dying from other diseases, those who die from tuberculosis usually show wide-spread lesions of the lungs.

1. *Miliary Tuberculosis of the Lungs.*—In nearly every case of pulmonary tuberculosis, miliary tubercles are found in some part of the lung, usually upon the surface and in the vicinity of some older process. Occa-

sionally, they are distributed throughout nearly the whole of the lung. In some places the lung, with the exception of the gray granulations, appears quite normal; in others, between the tubercles, the lesions of simple pneumonia are seen at various stages. There is also an acute bronchitis of the larger and smaller bronchi. The microscope shows that the tubercles develop in the walls of the small bronchi or in the alveoli. In gross appearance, the lungs in these cases resemble those of acute broncho-pneumonia, with the exception of the gray granulations on the surface and throughout the substance of the lungs. There are also gray granulations, and in most cases some nodules. The pleura is usually normal except where there are tubercles. This form of the disease represents a stage of tubercle bacilli throughout the lungs, the result of the inflammation excited by their presence.

2. Tuberculous Broncho-pneumonia.—This is the most characteristic form of tuberculosis in children, and it is the one which at this age usually predominates. In this form of the disease there are produced in the lungs, as in the larger caseous areas, some of which have usually become caseous at the time the case comes to autopsy. The process is usually of a somewhat subacute course. With the lesions met with in this form are associated those of simple broncho-pneumonia.

The pleura is involved in almost every case, and there are dense connective-tissue adhesions which bind the lung to the chest wall, the diaphragm, and the pericardium, or the lung is thickened and contains caseous deposits. Occasionally the nodules are large, but it is almost always sacculated and small.

Both lungs are usually involved, but one is more affected than the other. There are found large areas of caseation, which sometimes involve an entire lobe, but more often several lobes. These portions of the lung appear much like those in ordinary pneumonia. The upper lobes are more affected than the lower, and especially that part of the lower lobe of the lung, on account of its frequent association with the bronchial glands; the disease very often extends from the point to the middle lobe of the right, or the lower lobe of the left lung. On section the affected part of the lung contains caseous nodules varying in size from a pin's point to a large walnut. They are of a pale yellow colour, and resemble caseous nodules. They contain giant cells and are usually filled with softened containing yellow pus. There is never in any part of the lung a large caseous area; and never is there a diffuse caseation of almost an entire lobe.

times no spot of softening is seen even in these large areas, but in many cavities are present.

Softening and excavation represent the final stages of the process in tuberculous pneumonia. Softening usually begins in the centre of a caseous part, often at several points at the same time. Areas of excavation large enough to deserve the name of cavities were present in about



FIG. 202.—TUBERCULOUS PNEUMONIA. A vertical section through the middle of the right lung of a child thirteen months old. The greater part of the upper lobe is uniformly caseous—a diffuse tuberculous pneumonia; near the centre the commencement of a cavity is seen; below it has the appearance of a consolidation from simple pneumonia. The part of the lower lobe shown is normal.



FIG. 203.—CAVITY FROM BREAKING DOWN OF TUBERCULOUS PNEUMONIA. Another view of the same lung, the section being made very near the posterior border of the lung. The cavity occupies at this point nearly the whole of the upper lobe. At autopsy this cavity contained numerous loose caseous masses, the largest being the size of a marble. The lower lobe is normal. (For history, see Fig. 208.)

half of my autopsies upon tuberculous patients, two years old and under. They vary in size from a cherry to a hen's egg, and sometimes a much larger one is seen (Fig. 203). They are usually rather deeply seated, and are partially or entirely filled with caseous masses or pus, but very seldom perforate the pleura, causing pneumothorax or pyopneumothorax.

It is rare in a young child to find cavities such as are seen in older children or in process of softening once begun usually advanced of the patient.

The bronchial lymph nodes are in these cases tuberculous, and not only those at the root of the



FIG. 204.—PULMONARY TUBERCULOSIS, EXTENSIVE CASEATION OF BRONCHIAL GLANDS. *History.*—Coloured child, 2½ years, was feeble breathing and flatness, suggesting emphysema. The sputum made for bacilli, all negative. For temperature showed a regular daily range from 100°.

*Autopsy.*—Almost complete caseation of left lung. Out right lung were small tuberculous nodules and many very large and caseous, but none broken down; those at the root of the lung but the chain following the main lung itself.

is made, a chain of these tuberculous glands with larger bronchi for some distance into the lung one may be discovered which has softened and small bronchus.

Microscopical examination of these cheesy masses most frequently begin as tuberculous deposits in bronchi, either in the mucous membrane, the fibrous wall; sometimes, however, they begin in the wall of the bronchus. Cell proliferation takes place, separating the

blood-vessel, and partly or entirely obstructing its lumen. Softening may take place and the contents be discharged into the bronchus or blood-vessel. About this focus other changes of an inflammatory character occur, as a result of which each cheesy nodule is surrounded by a zone of simple broncho-pneumonia which tends, in a measure at least, to limit the tuberculous process. The larger caseous areas are formed by an extension of this process to the zone of pneumonia which surrounds it; but in its further growth it is still preceded by a simple pneumonia. The rapidity with which the lesions advance differs much in the different cases; in infants the progress is apt to be continuous until the death of the patient; in older children it is usually slower, and interrupted by intervals of arrest and even of partial retrogression.

Not infrequently one sees in the post-mortem room one or two caseous, or less frequently calcareous, nodules encapsulated by firm, organised connective tissue when a most careful search fails to show any other tuberculous lesion in the lung. If, however, the nodules are widely scattered through the lung, such an arrest of the process is not to be expected.

3. Chronic Pulmonary Tuberculosis, Chronic Phthisis.—In children who have passed the seventh or eighth year the pathological process resembles that seen in adults; but in younger children, and especially in infants, nothing corresponding to it is met with.

At this period the nearest approach to this condition is seen in the cases of tuberculous broncho-pneumonia, which run a slow, irregular, and somewhat chronic course. The essential features of the process in these patients is a chronic interstitial broncho-pneumonia with tuberculous nodules which rarely undergo softening, but usually become encapsulated.

The gross lesions closely resemble those of simple chronic broncho-pneumonia. There are the same generalised pleuritic adhesions and the shrunken cicatricial condition of the part of the lung most affected, with bronchiectasis, compensatory emphysema, etc. The tuberculous nodules are old and for the most part converted into dense fibrous tissue, in the centre of which, however, some softened, caseous areas are often seen.

*Bronchial Lymph Nodes (bronchial glands).*—The prominence of the lesions of the lymph nodes is one of the most striking features of tuberculosis in infancy and early childhood. Those which are most frequently affected are connected with the bronchi. The lymph nodes, to which the term "bronchial glands" is generally applied, consist of three groups: the first of which surrounds the trachea; the second is situated at the bifurcation of the trachea and surrounds the primary bronchi; while the third follows the course of the bronchi into the lung, being found, according to anatomists, as far as the fourth division. The anatomical relation of the different groups should be borne in mind, since upon them the symptoms principally depend. The first group, or the peri-tracheal

lymph nodes, are in relation with the superior vena cava, the pulmonary artery, the pneumogastric and recurrent laryngeal nerves; the second group, at the bifurcation of the trachea, with the œsophagus, pneumogastric nerve, and aorta; the third group, with the bronchi and branches of the bronchial and pulmonary arteries and veins.

All the groups are usually involved at the same time, but in various degrees, and in most cases those belonging to one lung to a greater extent than the other; in my own cases those of the right side have more often been involved than those of the left. There may be six or two or three tumours as large as a hazelnut, or there may be a mass of three inches in diameter, which is made up of ten to twenty of the glands fused together by inflammatory products, completely surrounding the trachea and both the large bronchi. It is rare that the individual glands are more than an inch in diameter, and most of them are smaller than this. A well-marked but not unusual example of this condition is shown in Plate XIX. There is usually found a chain of these tuberculous glands following the course of the large bronchi for some distance into the lung; sometimes these are almost as large as the external jugular gland (Fig. 204); at other times they are not noticed unless a somewhat careful dissection is made. The process is not infrequently more advanced in these deeply seated glands than in those situated at the root of the lung; and lesions here are also more important, as it is very frequent that the lung becomes involved.

The pathological changes through which these glands pass as a result of tuberculous infection are very similar to those already described with reference to the cervical glands. Suppuration is less frequent in the region of the neck, while calcific degeneration is much more common. This applies especially to children over three years old. In infancy suppuration is not infrequent in the bronchial glands, while at this age calcification is extremely rare. Although the process has gone on to caseation, these inflammatory products with bacilli may become encapsulated, and may remain innocuous for an indefinite period. The bacilli may die or may exist here, living, for years. At any time the old process may be lighted up, and a more or less rapid dissemination of tubercle bacilli take place through the lungs or through the whole body. Latent tuberculosis more frequently exists in the bronchial lymph nodes than in any other structure in the body.

Secondary lesions may be produced by these lymph nodes. The pneumogastric and recurrent laryngeal nerves may be surrounded by one of these cheesy masses which causes pressure and irritation. The œsophagus, the trachea, or the bronchi may be compressed or opened by ulceration. The superior vena cava usually suffers only compression, but this or one of the other large vessels may be opened. Ulceration may also take place into one of the large or small bronchi or the trachea. If the gland

PLATE XIX.



**TUBERCULOSIS OF THE TRACHEO-BRONCHIAL LYMPH NODES.**

From a fairly nourished child, four months old, who was under observation for three weeks, with slight fever and a most severe, teasing, dry cough, which was almost constant, and upon which no treatment seemed to have the slightest effect. At first there were no signs of disease in the lungs; later there were a few coarse scattered râles.

There were small tuberculous deposits throughout both lungs, with quite a large area of cheesy pneumonia in the right middle lobe, and scattered miliary tubercles in other organs.



softened and broken down, and if the bronchus is a small one, the only result of this may be a rapid spreading of tuberculous infection throughout the lung. If sudden rupture occurs, a large caseous mass may escape into the trachea, or a large bronchus, with a result similar to that produced by any other foreign body. If suppuration occurs, the abscess may rupture into the surrounding cellular tissue, causing mediastinal or retro-oesophageal abscess. This may open externally at the suprasternal notch, or in the first or second intercostal space, or may ulcerate into any of the large vessels, the oesophagus, or the pericardium.

*Pleura.*—This is rarely normal in any case of tuberculosis. In acute general tuberculosis the only lesion may be a deposit of miliary tubercles upon the visceral pleura. In most of the other cases there are found fibrous adhesions over the part of the lung involved, binding it to the pericardium, the diaphragm, or the chest wall. The amount of thickening of the pleura varies a good deal, but is rarely great. Pleurisy with a serous effusion is not common in infants or young children; when it occurs it is apt to be sacculated. Hæmorrhagic exudation is very rare at this age. Empyema is also rare, being seen in but five per cent of my cases, and then it was small and sacculated. Pneumothorax and pyopneumothorax are very rare in children under three years of age.

*Heart.*—It is exceptional for the pericardium to be affected even in the most generalised forms of acute miliary tuberculosis. In such cases the usual lesion is a deposit of a few gray tubercles upon the visceral surface. In chronic cases other lesions analogous to those of the pleura may be seen, but all are rare in childhood. In rare instances miliary tubercles are seen upon the endocardium.

*Brain.*—Tuberculosis of the brain is very common during infancy, being then associated in nearly all cases with general tuberculosis. Miliary tubercles are occasionally found in small numbers in cases which have presented no symptoms. The lesions of tuberculous meningitis have already been described. Cheesy nodules are rare in infancy, being noted in but 2.5 per cent of my own autopsies, which were mainly on children under three years old; while in the Pendlebury Hospital cases, including those between four and twelve years old, they were noted in 24.4 per cent. These nodules vary in size from a pea to a hen's egg; they are usually associated with tuberculous meningitis, but they may exist alone. When they are large they rank as cerebral tumours, being most frequently seen in the cerebellum.

*Liver.*—This is frequently involved in general tuberculosis, although it is doubtful if it is ever the seat of primary infection except in the congenital cases. Usually the only lesion is the presence of miliary tubercles on its surface and in its substance, and in most cases these are not numerous. They are found in about two-thirds of the cases. In a smaller number there are tuberculous nodules of various sizes, especially about

the biliary ducts. In nearly every protracted fatty. In very late cases of tuberculosis of the seat of amyloid degeneration.

*Spleen.*—This is more frequently affected lesions are similar. The size of the spleen is miliary tubercles are present; but with tube greatly enlarged. Amyloid degeneration is foci as in the liver.

*Stomach.*—Tuberculosis of the stomach in both its contents and its acid reaction seem infection from the mouth. Tuberculous ulcers autopsies, which is a larger proportion than in

*Intestines.*—That these are less seriously older children is rather surprising when we compare the intestines of infants to other forms of infection of this difference seems to be that intestinal secondary to disease of the lungs, primary lesions infants die from the more rapid tuberculous process before there has been time or opportunity for the importance to occur. The intestinal lesions enteric lymph nodes with which they are almost described elsewhere.

*Peritonæum.*—In early infancy the peritonæum even in general tuberculosis, and at this age it is the seat of the principal tuberculous process. It is frequent. In most cases of general tuberculosis of miliary tubercles; less frequently there are other inflammatory products. The lesions are described with Diseases of the Peritonæum.

*Thymus Gland.*—In five of my cases tuberculosis in the thymus gland, the size varying from a small size these were cases showing widely disseminated

*Pancreas.*—In four of my cases this organ contains tuberculous nodules, all of them being cases of

*Uro-genital Organs.*—Serious tuberculosis of the tract is very rare in children. Miliary tubercles are in about one-third of my autopsies on the organs are generally few in number. Large tubercles I have seen but once in a young child. The first was in the fourteenth year. In four of my autopsies tuberculosis was found in the suprarenal capsules. Tuberculosis is observed in rare instances among children.

Tuberculosis of the bones and of the external organs has already been described.

## THE CLINICAL FORMS OF TUBERCULOSIS.

**I. General Tuberculosis.**—Cases of tuberculosis present a wide variety in their symptomatology, depending upon the seat of infection, the rapidity with which the bacilli are disseminated through the body, or the numbers in which they enter. The general symptoms often precede the local ones, but are not recognised as those of tuberculosis. Often it is not suspected until the process is quite well advanced in some one organ.

**IN INFANTS.**—The early symptoms in infancy are often only those of failing nutrition. The patients are pale, thin, do not gain in weight no matter how fed, and finally lose steadily without sufficient reason. There may be no cough or fever sufficient to attract attention, and the case may even go on to a fatal termination without anything else than simple marasmus having been suspected, tuberculosis being first recognised at the autopsy.

More frequently, however, there are developed toward the end of the illness both the symptoms and signs of pulmonary disease and fever. These are generally found together, as the process in the lungs is usually the cause of the rise of temperature. The febrile symptoms are often not seen until the last two or three weeks of life. The course of the temperature is irregular. It is never of the hectic type and rarely high. The usual range is between 100° and 102° F. The pulmonary symptoms are generally few and not very well marked. There is some cough, but it is rarely severe. The breathing is more rapid than would be explained by the temperature alone. Severe dyspnoea and cyanosis are rare, and are seen only at the close of the disease. The physical signs are those of either localised or general bronchitis. Digestive symptoms are usually present late in the disease, but they are rarely due to a tuberculous lesion of the stomach or intestines.

The progress of the case after constitutional symptoms develop is usually steadily downward, and the child lives but a few weeks at most. Death generally occurs from progressive asthenia without the development of any new symptoms, or cerebral symptoms rapidly develop, and the child is carried off in a few days by tuberculous meningitis. Sometimes there is a rapid spreading of the disease in the lungs, and death occurs with symptoms of acute pneumonia.

General tuberculosis in infants is to be differentiated principally from marasmus with bronchitis; less frequently it may be confounded with hereditary syphilis.

**IN OLDER CHILDREN.**—The development of active general tuberculosis in older children is usually preceded by a protracted period of indefinite symptoms. They are persistently anæmic without evident reason; they lose weight; digestion is disturbed; the appetite is capricious.

cious; they sleep badly; they are irritable, fitful. These symptoms indicate only a gradual decline, but may readily be explained by many other causes. They should, however, excite a suspicion of tuberculosis if the surroundings or inheritance is predisposed to it.

After these indefinite symptoms have lasted some time, sometimes the prodromal symptoms of fever and cough are the first evident symptoms. In some cases progress rapidly to a fatal termination. In the majority, however, the disease runs a chronic course. The temperature is not often high, and the rose-coloured spots are not present. The aspect of the patient is strikingly suggestive of tuberculosis. The course of the temperature and the duration of the fever have to deal with some other condition.

After the fever has lasted from one to three weeks, signs of localised tuberculosis, generally in the lungs, appear. The fever declines gradually, and although the patient feels well. He is still weak and does not gain in weight. The disease may grow rapidly worse and the course of the fever becomes irregular, with alternate exacerbations and remissions. Inexplicable fever sometimes puzzles the physician for weeks before the characteristic features which are present. Before very long wasting may not be rapid, but is progressive. The process is usually unmistakable; but in most of the cases it does not advance far enough in some one of the organs to give signs or symptoms before he can be sure of the disease. Out of five this is in the lungs, and frequently the sputum may reveal the bacilli. Less often in the peritonæum, the brain, or a general infection of the body. If in the lungs, the process manifests itself as pneumonia whose tuberculous character may soon be evident from its location—the apex or the middle of the lung. From the fact that the general symptoms, fever and cough, preceded the local signs. From this time, the disease is typical tuberculous broncho-pneumonia.

If the tuberculous process is localised in the meninges, headache, drowsiness, irregular pulse, finally convulsions and coma—in short, the meningitis; if in the peritonæum, there are gas or fluid, tenderness, pain, diarrhoea, or co

glands, there is a general enlargement of those situated externally, sometimes with symptoms indicating similar changes in those at the root of the lung.

**II. Pulmonary Tuberculosis.**—Tuberculosis of the lungs in children may be seen in a variety of clinical forms which correspond with the different pathological conditions. The pathological conditions are often associated, yet the main clinical types are sufficiently distinct to give quite a definite picture. These types are: (1) miliary tuberculosis of the lungs; (2) bronchitis with small, scattered, tuberculous nodules; (3) tuberculous broncho-pneumonia with areas of consolidation, often extensive, which may be followed by caseation and excavation, or by chronic fibrous induration.

**MILIARY TUBERCULOSIS OF THE LUNGS.**—This is not a common form of pulmonary tuberculosis, but may be met with even in young infants.



**FIG. 205.—MILIARY TUBERCULOSIS OF THE LUNGS.** Infant fourteen months old; symptoms of marasmus; no elevation of temperature; tuberculides of the skin; positive von Pirquet reaction; no pulmonary signs or symptoms. The radiograph shows great numbers of small tuberculous deposits scattered through both lungs.

Both the general and pulmonary symptoms and the physical signs are rather obscure and indefinite, and often the diagnosis is not made. Occasionally the only symptoms are those of marasmus, neither fever nor physical signs in the chest being present (Fig. 205). As I have seen it in young children, it has seldom been attended by high temperature,

101° to 103° F. being the usual range. Throughout the greater part of the disease it is often lower than this, and toward the close perhaps rather higher. It is not a hectic type of fever, and it seldom touches the normal line.

The duration of the disease in these cases, after fairly definite symptoms begin, varies from ten days to a month. At first, and often for two or three weeks, the temperature is almost the only symptom. Cough is slight, inconstant, and seldom loose. There is no sputum. The respirations are only moderately accelerated, in many cases not enough to draw attention to the lungs as the seat of disease. There is no rapid wasting, the loss in weight being usually not more than would be expected with any other febrile disease. None of the other symptoms suggest tuberculosis. The usual problem in diagnosis is to discover the cause of the fever. Often the most careful examinations of the chest made daily reveal nothing more than a few scattered râles. These change in position from time to time, and it frequently happens that for days none are heard. After the disease has progressed somewhat further, the liver and spleen are generally enlarged. Cerebral symptoms may develop, and the case terminate as tuberculous meningitis, but more often it is the pulmonary symptoms which are dominant. The respirations become more rapid; the cough is frequent, but rarely loose; there may be attacks of cyanosis. Still the only definite signs are the râles, now fine and moist, and diffused generally over the chest. The case usually ends in death by exhaustion, but without rapid or marked wasting. One of the most striking things in the clinical picture is the disproportion between the severity of the general and pulmonary symptoms and the few physical signs in the chest.

**TUBERCULOUS BRONCHITIS.**—This is not an infrequent condition even in infancy. In many, perhaps in most, cases it marks the earliest clinical stage of a tuberculous broncho-pneumonia, but this is not always true. The condition seems, therefore, of sufficient importance to require separate consideration. Besides bronchitis, there are found at autopsy a few small tuberculous nodules, and tuberculosis of the bronchial glands, although these may give neither signs nor symptoms during life. The symptoms of this condition are few and not distinctive, and may differ in no respect from bronchitis due to other causes. Tuberculosis may not even be suspected until the lesion has so far developed as to be classed as tuberculous broncho-pneumonia. Cough is present, but has nothing characteristic about it except its persistence. Fever may be absent for a long time, but comes as the disease advances. Then it is low and very irregular, the temperature generally varying from 99° to 101.5° F. There may be slow but progressive loss in weight, or the infant may gain regularly for a number of weeks in spite of the cough. This fact often leads to a mistake in diagnosis. The nutrition is influenced much

more by the condition of the digestive organs than by the tuberculous process. Other symptoms generally regarded as belonging to early tuberculosis, such as pallor, anæmia, perspiration, etc., are usually absent. The physical signs are few and not characteristic. Scattered râles, sometimes coarse and sometimes finer, but inconstant, are all the signs that are present for a long time, often several weeks.

Cases like these are recognised as tuberculous only by finding bacilli in the sputum or by one of the tuberculin tests. It has been my custom to consider as probably tuberculous every infant who has been for any length of time in contact with a tuberculous parent or other member of a household. Regarding all such infants as suspicious has led me in hospital practice to search the sputum carefully for bacilli, with the result of finding them, sometimes in great numbers, in infants whose only outward symptom was a moderate cough, and who were admitted to the hospital for some other reason. At other times the condition has been unexpectedly discovered by making routine eye or skin tests in hospital inmates with tuberculin. A typical reaction having been obtained in a child not hitherto suspected, the diagnosis of tuberculosis has been subsequently confirmed by finding bacilli in the sputum, although the only signs in the chest were a few indefinite râles and the only outward symptom a moderate cough. How many infants there are with such a form of tuberculosis and how long such a condition may continue without more definite signs developing, one can only conjecture; but the number of such cases is, I am convinced, not small. They form a very distinct but important group of tuberculous cases. The regularity with which bacilli are present in the sputum indicates what a factor they may be in spreading the disease. How many recover and in how many the disease goes on to the development of more serious lesions it is impossible to say.

**TUBERCULOUS BRONCHIO-PNEUMONIA.**—This is altogether the most frequent form of tuberculosis seen in young children. It may be primary in the lungs or it may be secondary to tuberculosis elsewhere, most frequently in the bronchial glands. It may be preceded by constitutional symptoms such as those described under the head of general tuberculosis. It may follow single or repeated attacks of what was apparently a simple acute bronchitis or broncho-pneumonia, whether it occurred as a primary disease or was in turn a sequel to one of the infectious diseases, especially measles, whooping-cough, or influenza.

Tuberculous broncho-pneumonia, as a rule, begins more gradually, and its course is less rapid than simple broncho-pneumonia, its progress being generally marked by weeks. When primary it is often preceded by symptoms described as tuberculous bronchitis. When it follows one of the infectious diseases it is usually engrafted upon the original disease without any intervening symptoms. The early symptoms are cough,

rapid respiration, fever, progressive weakness, and anæmia. The weight may be at first stationary, but soon there is steady loss, which may continue until there is marked emaciation. At first the usual range of temperature is from 100° to 102° F.; later it is rather higher than this. In many of the cases it differs little from the temperature of simple broncho-pneumonia. Sometimes the general symptoms are severe and the physical signs wide-spread, and yet the range of temperature is not high. To be sure, this is occasionally seen in simple broncho-pneumonia, but it is more frequent in tuberculosis. The cough early in the disease is slight, but later becomes severe and often distressing. In infants and young children it may be of a paroxysmal character, resembling pertussis. Expectoration is not often seen in those under five years old. Bloody expectoration is very rare in children.

The conditions in the lungs which give physical signs are bronchitis of the smaller tubes with areas of complete or partial consolidation. In character, these signs are identical with those of simple broncho-pneumonia. They may be scattered throughout the whole of both lungs; but when localised they are more frequently in the upper than in the lower lobes, and more frequently in front than behind. Although both lungs are involved, they are usually not affected to the same degree. The patient may die before signs of complete consolidation are present; more often there are during the last few days areas of consolidation, as shown by bronchial breathing and voice and dulness. In some cases although wide-spread lesions are found at autopsy the physical signs during life are few and indefinite; sometimes there may be almost none. (See Fig. 205.)

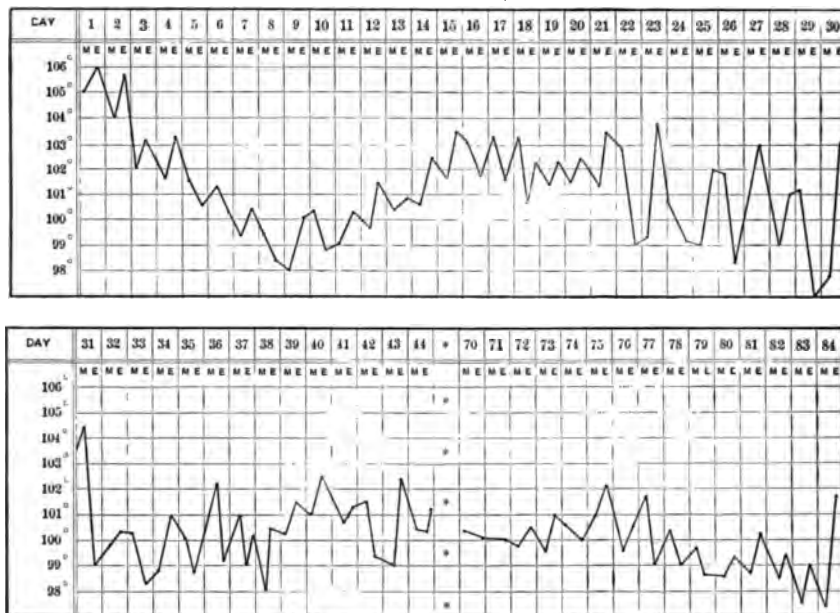
From the beginning of acute symptoms the progress of the disease is steadily downward, death occurring as in simple broncho-pneumonia. The end is marked by cyanosis, great dyspnoea, weak pulse, and extreme prostration. In a few cases there develop shortly before death cerebral symptoms, indicating tuberculous disease of the brain. Such symptoms may be the first to lead the physician to suspect the process to be a tuberculous one. But even this is not conclusive, for one may be dealing with an acute meningitis due to the pneumococcus. Lumbar puncture will decide.

In the more protracted cases there are found in the lungs caseous nodules, with larger areas of caseous pneumonia, and usually some areas of softening. The process is not usually so generalised as in the cases just described, but as in them there is always associated a certain amount of simple pneumonia. The pathological process may terminate (1) in diffuse caseation, or (2) in localised caseation and excavation, or (3) in partial resolution and the development of a chronic fibroid pneumonia. In the first two varieties the progress is as a rule steadily downward to a fatal termination, which takes place in from one to three



months. In the third form, which is described later, there is partial recovery.

The mode of onset will depend upon the conditions under which the disease develops. When the general symptoms of tuberculosis have preceded those in the lungs, the evolution of the latter is gradual, with cough, rapid breathing, dyspnoea, increased prostration, etc. When the pulmonary symptoms are present from the beginning, they are the same as in simple broncho-pneumonia, with the exception that they usually come on less acutely. The latter is true of cases which are secondary to some other form of tuberculosis in the bones, peritonæum, etc.



**FIG. 206.—TUBERCULOSIS FOLLOWING MEASLES.** Child sixteen months old, inmate of an institution. Chart begins on fifth day of a severe, but uncomplicated attack of measles, and shows a natural decline to normal. Fever then returned and continued till death, twelve weeks later. Record for the period which is omitted was much like that which immediately precedes and follows. Early symptoms not acute, only slow wasting, slight cough and fever, with scattered râles throughout chest. Signs of consolidation not distinct till eighth week, then present in right upper lobe. Toward the end, rapid emaciation, marked pulmonary symptoms, and signs of cavity at right apex. Autopsy showed a large cavity, extensive tuberculous deposits throughout both lungs and in nearly all abdominal organs.

When pulmonary tuberculosis follows measles (Fig. 206) or whooping-cough which has been complicated by simple pneumonia, the early symptoms may present no unusual features. After two or three weeks the temperature gradually falls, and the physical signs improve, but neither quite disappears. The cough continues, though its severity somewhat abates. In the course of a few weeks the child, who has meanwhile

improved somewhat in his general condition, becomes distinctly worse, often without any assignable cause. The temperature rises to  $102^{\circ}$  or  $103^{\circ}$  F.; the cough increases, and an extension of the disease in the lungs is evident by the physical signs. In other cases the progress of the disease, after a pneumonia which complicates measles, is without an intervening period of apparent improvement. It sometimes happens that the attack of measles or whooping-cough is not accompanied by any serious pulmonary symptoms, and the case goes on to apparent recovery, except that there remain anæmia, a slight cough, and fever. The temperature, although not high, persists; but it may be two or three weeks before there are present definite symptoms and signs of disease in the lungs.

Fever is a constant accompaniment of all active tuberculous processes in the lungs in the child as in the adult, it being absent only during the periods of remission which occur in the cases of slow and irregular progress. It is a very important guide to the progress of the disease. The early fever may depend in part upon coexisting broncho-pneumonia, and its course may resemble that of simple pneumonia of the protracted variety. There is no typical curve. The fever is not often steadily high, and in many cases it is never high (Fig. 207). It frequently runs for

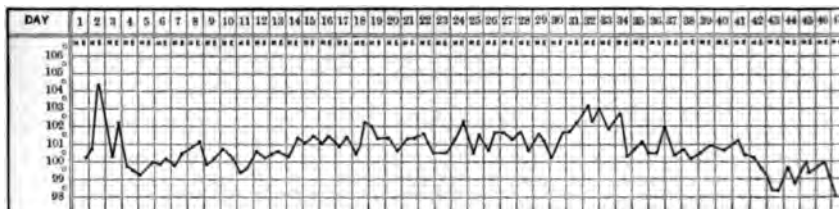


FIG. 207.—TUBERCULOUS PNEUMONIA; GENERAL TUBERCULOSIS. Patient eleven months old, and under observation at the time he was taken sick. Chart of entire illness is given. Disease began as an acute pneumonia in lower part of left axilla and spread to entire lower lobe. Early signs of consolidation; at end of two weeks, flatness so marked that a needle was inserted, fluid being suspected. Vomited frequently, and had loose discharges from bowels throughout the illness; abdomen much swollen for last two weeks. Autopsy showed cheesy pneumonia of part of the upper and the entire left lower lobe, where there were two small cavities. Recent tubercles found throughout right lung, and extensive deposits in abdominal organs with peritonitis, and intestinal ulcers.

several days between  $99^{\circ}$  and  $102^{\circ}$  F., and then, without evident cause, rises to  $104^{\circ}$  F. or over. In infants the morning temperature is frequently subnormal, although the evening temperature may be  $102^{\circ}$  or  $103^{\circ}$  F. Even toward the close of the disease, when softening and breaking down are actively going on, the regular hectic temperature of adults is rarely seen in a young child (Fig. 208). While the presence of fever is of great significance, its course has almost no diagnostic importance in early life. Especially should one beware of drawing the conclusion



months old, where the hæmorrhage was due to the rupture of an aneurism in the wall of a cavity. Herz, in 247 clinical cases of tuberculosis in children, records 8 of hæmoptysis—4 of them under five years, and the youngest only eighteen months old. The records of 131 autopsies on tuberculous children in the Pendlebury Hospital show that hæmoptysis was four times a cause of death; two of these patients were under five years, and one was only twelve months old. I have never met with a case of hæmoptysis under five years old.

The respiration is accelerated, and usually out of proportion to the rise in temperature. As the lung becomes more and more extensively invaded there is constant dyspnœa. The pulse is rapid in the early stage, and continues so throughout the disease; toward the end it becomes weak and irregular. Irregular respiration and a slow, irregular pulse may occur at any time from the development of cerebral complications.

Pleuritic pains in the chest are not frequent in children. Gastro-intestinal symptoms, such as indigestion, vomiting, diarrhœa, etc., are generally present, but are not peculiar to this disease. They usually depend upon the patient's general condition, only exceptionally upon tuberculous disease of the stomach or intestines. The characteristic symptoms of intestinal tuberculosis—abdominal pain, tenderness, uncontrollable diarrhœa, and intestinal hæmorrhage—are seldom met with in children under five years. I have seen but two cases. With such symptoms, and sometimes when they are doubtful or absent, careful palpation of the abdomen may disclose the presence of enlarged mesenteric glands. When these are not readily felt through the abdominal walls, they may sometimes be discovered by a rectal examination.

The spleen is often enlarged, sometimes very much so, but this does not occur with sufficient frequency to be of much diagnostic value. It may be due to tuberculous deposits, to causes connected with the lungs or heart, or to fever. The liver is not enlarged from tuberculous deposits, but may be so from amyloid or fatty degeneration, or from obstructed circulation, as in the case of the spleen.

Dropsy is rare. It may depend upon anæmia, upon complicating nephritis, especially amyloid degeneration, upon cardiac or pulmonary conditions leading to interference with the return circulation, or upon pressure of tuberculous retro-peritoneal or mesenteric glands upon the inferior vena cava. Clubbing of the fingers is occasionally seen in cases running a very protracted course, and is due to obstructed circulation.

Anæmia is commonly associated with wasting, and it is of special importance when the latter is slight or absent. It is a frequent sequel of acute disease in infancy when not dependent on tuberculosis; when, however, it is associated with low fever, cough, and persistence of râles in the chest, it should excite apprehension.

**CHRONIC TUBERCULOUS PNEUMONIA.**—In young children this is a chronic interstitial pneumonia associated with tuberculous deposits. These cases have usually had their beginning in one of the acute forms. There is a slow convalescence and apparent recovery, although this is not complete. Often a slight cough remains, or returns from the slightest exposure or other exciting cause. The child does not regain his former weight or vigour, and careful examination of the lungs shows that some abnormal signs remain.

After a few months, possibly, the child has another attack resembling the first. It is accompanied by fever, cough, and perhaps there is a fresh consolidation of some part of the lung, generally in the neighbourhood of the old disease. All active symptoms finally subside, and most of the signs of recent disease disappear; but it is then usually found that the condition of the lung is not quite so good as before this second illness. The acute attacks may be repeated several times and pass under the name of bronchitis, broncho-pneumonia, or pleurisy. They may extend over a period of years. The general health in the interval is not good, there being present in most cases anæmia, with the usual symptoms of malnutrition; the children are regarded as very delicate.

The course of this disease thus differs in no essential particulars from that of simple chronic broncho-pneumonia; the physical signs likewise are identical in character, although they may differ in their location. They are generally found in the same conditions as are the signs in the more rapid forms of pulmonary tuberculosis in early childhood. A fatal result in these cases is usually brought about by the development of acute tuberculous pneumonia or miliary tuberculosis of the lungs, by tuberculous meningitis, or by a simple broncho-pneumonia.

**PHYSICAL SIGNS OF PULMONARY TUBERCULOSIS.**—Speaking generally, except in situation there is little difference in a young child between the signs of a bronchitis or broncho-pneumonia due to the tubercle bacillus, and those of the same lesions when due to other causes. Cavities, although present at autopsy in most of the advanced cases, are seldom of such size or so situated as to be recognised during life. In children over six or seven years old, the signs are essentially like those in adults.

The upper lobes are the seat of the most advanced disease twice as frequently as the lower lobes, and the right lung rather more frequently than the left. The region most often involved is the middle zone of the lung. If the signs appear first behind they are usually in the interscapular space; if in the lateral part of the chest, they are in the middle or upper part of the axilla; if in front, they are in the mammary region. The explanation is found in the fact that the disease in infants and young children so often extends from the lymph nodes at the root of the lung to the lung itself. The physical signs themselves may be grouped under four heads, corresponding to the pathological conditions existing

in the disease, viz., (1) bronchitis; (2) partial consolidation; (3) complete consolidation; (4) excavation. The early signs are almost identical with those described in broncho-pneumonia. As a rule, however, the transition of the signs from one stage to another is much slower in tuberculous than in simple broncho-pneumonia.

Tuberculous bronchitis gives râles which may be of all sizes and varieties, localised or general. If the process goes on to a partial consolidation there are gradually developed in addition slightly impaired resonance or even dulness, broncho-vesicular respiration, and increased voice. These signs are usually over a localised area. Later the signs of complete consolidation are present—marked dulness, increased fremitus, bronchial respiration, and voice, but still râles and friction sounds are generally heard.

The later signs depend upon what course the pathological process follows. If it terminates in a diffuse or localised caseation, the signs differ little from those of a lobar pneumonia with extensive and complete consolidation except that the dulness on percussion is usually greater. There may be even flatness, so marked as to suggest the presence of a pleural effusion. Empyema is often the diagnosis made. These signs may persist until the death of the patient from exhaustion.

If the caseation is localised and followed by excavation, the signs of a cavity may be present. Cavities, however, are often so small and deeply seated as not to give definite physical signs. Furthermore, they are frequently filled with thick pus or cheesy matter, and rarely communicate freely with the bronchi. If large and superficial they give the same signs as in adults. Like the areas of tuberculous pneumonia, they are most frequent in the middle zone of the lung in front. In the young child similar signs are often present where there are only dilated bronchi associated with a fibroid condition, or when a superficial bronchus is surrounded by an area of diffuse caseation. Cavities are very often diagnosed when they do not exist, and quite as often overlooked when present.

If the acute process terminates in a chronic tuberculous pneumonia the signs are those of an unresolved or slowly resolving pneumonia, in which the area of consolidation gradually diminishes, but the signs do not altogether disappear. When recovery goes further there may remain only some dulness on percussion, broncho-vesicular respiration, râles, and friction sounds. Such signs may last indefinitely, exacerbations and remissions occurring from time to time. These signs can not be distinguished from those of simple chronic broncho-pneumonia.

DIAGNOSIS OF PULMONARY TUBERCULOSIS.—In arriving at a diagnosis one should investigate the family history, surroundings, and previous condition of the patient; also consider the mode of onset, the course of the disease, and the evidence afforded by the examination.

A careful examination of the family history and surroundings should be made to determine the existence of pulmonary tuberculosis in the parents or in other members of the household. Inquiry should also be made regarding meningitis, disease of the cervical glands, spine, hip, knee, or ankle, especially in other children of the family. Other conditions favourable for acquiring the disease should be considered, as in cases where a child has been reared in a tenement house, or has been long an inmate of a hospital or other institution. In the child's previous history, it is important to know if he has had measles or pertussis, and whether they were severe, accompanied by pulmonary complications, or followed by a protracted cough or obscure fever. The child's general constitution should be considered, whether he is delicate, narrow-chested, poorly nourished, or habitually anæmic.

In its symptoms and course it is with simple broncho-pneumonia that tuberculous disease is likely to be confounded. The onset of simple pneumonia is usually rapid and often abrupt; tuberculous pneumonia usually develops gradually with constitutional symptoms preceding the local ones by several days or even weeks. In acute tuberculosis one is often struck by the disproportion between the general symptoms—loss of flesh, prostration, and temperature—and the local evidences of pulmonary disease. When the pulmonary disease lasts longer than usual the question arises whether we have to deal with a case of persistent broncho-pneumonia or with tuberculosis. In children whose general condition is poor it is not infrequent for simple broncho-pneumonia to resolve slowly or to go on to the development of chronic interstitial pneumonia, so that other means of diagnosis are needed.

The course of the temperature can not be depended upon to differentiate any form of pulmonary tuberculosis from simple broncho-pneumonia. Anæmia and wasting are usually more marked in tuberculosis, and in most cases they are progressive. A high leucocyte count, e. g., above 20,000—especially when accompanied by a high polymorphonuclear percentage, strongly favours pneumonia. Meningitis developing during a pulmonary disease of doubtful character is generally tuberculous, and its occurrence is usually to be interpreted as establishing the tuberculous nature of the process in the lungs. But acute pneumococcus meningitis may occur under very similar circumstances, and only a lumbar puncture may differentiate between them. A copious mucopurulent expectoration is seen quite as frequently in the other forms of chronic pneumonia as in the tuberculous variety.

*Examination for Bacilli.*—Discovery of the bacilli in the sputum of even young infants is by no means impossible, nor even a very difficult matter. Both time and patience are required, and in most cases repeated examinations are necessary. Infants do not expectorate, but cough up the bronchial secretion into the pharynx and swallow it. Sputum must

therefore be obtained from the pharynx or the œsophagus; to seek for the bacilli in the vomitus, as has been recommended, is almost a hopeless task. The method which has given me the most satisfactory results is to excite a cough by irritating the pharynx, and then to catch the sputum brought up into view upon a cotton swab or a bit of muslin in the jaws of an artery clamp. Inversion during a paroxysm of coughing sometimes causes the infant to discharge a considerable mass of muco-pus into a sputum cup. By the procedure mentioned it has not been found more difficult to obtain good sputum for examination in very young patients than in adults. Good sputum may be described as muco-purulent masses, for bacilli are very seldom to be found in clear, glairy mucus. Following the method described, bacilli have been found in over eighty per cent of my hospital cases of pulmonary tuberculosis in infants, although in over half of them the disease was not advanced, judging by symptoms and physical signs.

Bacilli may readily be found in the stools of many children suffering from tuberculosis. Their presence does not necessarily indicate a tuberculous lesion of the intestines, for their source is more frequently a pulmonary lesion, the bacilli being coughed up and swallowed. Hence, it is sometimes easier to find them in the stools than in the sputum. They must be carefully differentiated from the smegma bacilli.

**III. Chronic Phthisis.**—This form of tuberculosis, with its chronic hectic fever, slow cavity formation, progressive emaciation, night sweats, etc., is very rarely seen before the fifth year, and it is not at all frequent until the tenth or twelfth year. In its symptoms, course, termination, and physical signs, it resembles the same disease in adults, and need not be described at length here.

**IV. Tuberculosis of the Bronchial Lymph Nodes (Bronchial Glands).**—This condition is usually associated with some form of pulmonary tuberculosis, but it may exist as the most important and sometimes as the only tuberculous lesion.

Its symptoms are usually associated with those of pulmonary or general tuberculosis; but they may occur when the pulmonary changes are too few to be recognised either by symptoms or physical signs. From the great frequency with which this lesion is found in infants and young children, it might be expected that local symptoms would be common in such patients. They are, however, in my experience, quite exceptional. Most of the cases in which well-marked symptoms occur are in children over two years old, and it is between the third and tenth years that they are usually seen. In infancy, although these glands are almost invariably affected, death in the great majority of cases occurs from the pulmonary disease, before the later changes in the glands have had time to develop.

General symptoms may or may not precede the local ones. The



latter are chiefly mechanical, and depend upon the size of the glands and upon their anatomical relations, and very little or not at all upon the nature of the changes in them. The most important relations, so far as the production of symptoms is concerned, are those which they bear to the pneumogastric and recurrent laryngeal nerves, the superior vena cava, the trachea, and bronchi; those less important are to the aorta, pulmonary artery, and œsophagus.

Pressure upon or irritation of the pneumogastric or recurrent nerves produces cough, dyspnœa, and sometimes a change in the voice. The cough is hoarse, persistent, and teasing, and frequently occurs in paroxysms which in many respects resemble those of pertussis, but it lacks the characteristic whoop, and is not accompanied by the expectoration of a mass of tenacious mucus. These paroxysms are severe and often prolonged, but careful observation shows distinct differences from those of pertussis, though by an unfamiliar ear the two are easily confounded. The dyspnœa, like the cough, is paroxysmal, and sometimes strongly resembles ordinary spasmodic croup; at other times it is like a severe attack of asthma. Such symptoms may come and go, but they are frequently prolonged, and usually in the interval between the severe seizures the patient is not wholly free from dyspnœa. Although the chief cause of dyspnœa is no doubt nerve irritation, it may be due in part to pressure upon the trachea or one of the large bronchi. In dyspnœa from pressure on the trachea the head is usually thrown back, and the obstruction is more frequently on expiration than on inspiration.

After such symptoms as those mentioned have existed for a few days or weeks, and in some cases without any warning, there may occur a sudden attack of asphyxia which may prove fatal. This is generally due to ulceration of a caseous gland into the trachea or a large bronchus and the escape of a large mass into the air passages, where it produces the same effects as does any other foreign body.

Of fifteen cases of this kind collected by Loeb, death by suffocation occurred in most in from five to ten minutes after the first definite symptoms; in some the fatal attack was preceded for some time by milder attacks or by a cough; in others no previous symptoms were present, the child being apparently in perfect health. Rarely after ulceration into the trachea the patient has recovered after coughing up a large amount of foul pus.

Pressure upon the superior vena cava is usually associated with spasmodic dyspnœa and cough, and causes cyanosis of the face and blueness of the lips. There is frequently a puffiness of the face, and there may be marked œdema. The coexistence of cyanosis with such œdema, when the urine is free from signs of renal disease, should always lead one to suspect pressure at the root of the lung. In some rare cases the interference with the return circulation has been so marked that meningeal

hæmorrhage has resulted. By a process of ulceration set up by these glands they may open, not only into the air passages, but into the pericardium, the œsophagus, or any of the large vessels. The last mentioned is usually followed by instant death. Aldibert reports two cases in which the pulmonary artery was opened, death occurring from hæmoptysis, as there was also a communication with one of the large bronchi. In Vogel's case the subclavian vein was perforated, and death resulted from the entrance of air. If ulceration takes place into the surrounding connective tissue, a mediastinal abscess may result, producing any of the pressure symptoms noted above, and, in addition, dysphagia from pressure on the œsophagus. Such an abscess may point in the suprasternal notch; it may open through the chest anteriorly between the ribs or at the xiphoid cartilage; or it may burrow along the œsophagus to the peritoneal cavity. As a rule, however, patients die of general tuberculosis before the local conditions have advanced so far.

**PHYSICAL SIGNS.**—In order to produce physical signs, the mass of tuberculous lymph nodes must be large enough to form a mediastinal tumour, or so situated as to produce pressure on the trachea or bronchi. As a rule, the signs are more characteristic behind than in front. Percussion may give dulness anteriorly over the first piece of the sternum but very rarely posteriorly; when present it is found along one or both sides of the spine from the third to the seventh dorsal vertebra. Auscultation posteriorly gives in the most marked cases a voice and respiration of a peculiar character, somewhat amphoric, but with a distinctly nasal quality. The auscultatory signs may so resemble those of a cavity that it is often difficult to believe that a cavity does not exist. If one of the primary bronchi or one of its lobar divisions is compressed, there may be very feeble respiration over one lung or one lobe; if the pressure is sufficient to prevent the entrance of air, or if one of these large tubes has been plugged by a caseous mass, there is an absence of respiratory murmur over a single lobe or an entire lung. This sign is of great diagnostic value, but it is not often present.

**DIAGNOSIS.**—Enlargement of the bronchial glands to a sufficient degree to produce symptoms, may occur in syphilis, in Hodgkin's disease, and in various forms of malignant disease of the mediastinum. A certain amount of swelling is seen in nearly all cases of simple bronchitis or pneumonia, especially in those running a subacute or chronic course. Whether this simple hyperplasia is ever sufficient to cause such symptoms as those mentioned is exceedingly doubtful. I have myself never known it to produce anything more marked than a spasmodic cough. The great infrequency of other forms of enlargement sufficient to be of any clinical importance, usually warrants us, from the symptoms mentioned, in making the diagnosis of tuberculosis. The development in a child of a chronic abscess in the anterior mediastinum, is almost always due to

tuberculous glands; and so is one in the posterior mediastinum, provided Pott's disease can be excluded.

The most important points for diagnosis are the association of a spasmodic cough with paroxysms of dyspnoea resembling asthma or croup, and œdema or congestion of the face. More stress is to be laid upon the symptoms than upon the physical signs; the latter are at most only confirmatory. The chief difficulty in diagnosis is found in those cases which present few or no other signs of tuberculosis, and which come first under observation with attacks of dyspnoea or asphyxia resembling those seen in laryngeal stenosis. In many such cases tracheotomy has been done without finding any cause for the dyspnoea, the autopsy showing it to be due to the ulceration and impaction of a caseous gland.



FIG. 209.—TUBERCULOUS BRONCHIAL GLANDS.

A very large mass upon the right side, A, A; a smaller one upon the left side, B, B.

In many cases very positive information is given by the X-ray, the radiographic shadows usually showing better on the right side than on the left on account of the heart (see Fig. 209). This means of diagnosis is, however, of no value in distinguishing tuberculous glands from enlarged glands due to other causes; the latter, however, are very rare.

**The Tuberculin Tests.**—*The Fever Reaction Following Tuberculin Injections.*—This is quite as reliable in children as in older patients. It is limited in its application, since most cases of active tuberculosis at this period of life are accompanied by fever. Since the other tests are

easier to employ and not open to the same objections, there is now very seldom a need for the use of this test.

*The Ophthalmic Test (Calmette or Wolff-Eisner Test).*—This generally gives reliable results, but its use is attended by some risk and it has no advantages over the von Pirquet test.

*The Cutaneous Test (von Pirquet's Test).*—Usually the forearm is the part chosen for inoculation. The skin is carefully washed with alcohol or ether. A small drop of pure tuberculin is placed upon the skin. With a small instrument resembling a tiny chisel a simple scarification for control is made at a distance of two or three inches from this drop. A similar scarification is then made through the drop. Linear scratches one-quarter inch in length with a sterile needle serve equally well as a means of inoculation and control. The child should be watched, and if very young the arm should be held until the skin is quite dry to prevent infection by rubbing. As an added precaution it may be covered with a piece of sterile gauze. The reaction consists in a red areola about the point or along the line of inoculation. This generally begins in from twelve to eighteen hours, rarely as late as twenty-four hours, and reaches its height during the next twenty-four hours. The diameter of the areola indicates the degree of reaction. It continues in most cases for from one to three days and slowly fades, often being followed by a slight local desquamation. Rarely there may be vesiculation. There is in most of the cases slight infiltration of the skin readily appreciable to the touch; and there may be a distinct induration. The more marked reactions continue for from four to ten days. Any definite inflammatory reaction which follows this course may be regarded as positive. The arm should be observed daily to note the results. There seems to be no relation between the intensity of the reaction and the extent or the activity of the tuberculous disease.

*The Puncture Test (Stich-reaction of Hamburger).*—There is injected just beneath the skin of the forearm a measured dose, from  $\frac{1}{100}$  to  $\frac{1}{1000}$  mgr. of tuberculin. The reaction is seen at two points; the greater, corresponding to the place where the fluid is deposited, the less, where the needle perforates the skin. Swelling, redness, induration and local rise of temperature are present. The reaction begins within the first twenty-four hours; the induration and discolouration of the skin last five to six days and slight desquamation follows. A reaction beginning later than twenty-four hours is not diagnostic. Hamburger's statement that in older children this is the most sensitive of all tests seems probable.

*Inunction Test of Moro.*—There is used for this test tuberculin made up with anhydrous lanolin, fifty per cent strength. A mass of this, the size of a pea, is rubbed for half a minute into the skin of the abdomen or chest over an area two inches in diameter. The reaction consists in the formation of a papular, sometimes a vesicular, eruption which ap-

pears, according to the severity of the reaction, in from twelve to forty-eight hours. It remains for several days and slowly disappears, being followed by pigmentation in the severer cases.

*A Comparison of the Different Tests.*—No one of the tests is so absolutely conclusive as is the demonstration of the tubercle bacillus in the sputum, the cerebro-spinal fluid, or elsewhere. One should not therefore depend upon local tests and omit the search for bacilli, even though it involves greater labour. While these tests when followed by a positive reaction furnish evidence of the existence of a tuberculous lesion, they do not enable us to distinguish between latent and active conditions. Thus, a child may give a positive skin reaction when suffering from acute pulmonary disease, which by its course is shown to be non-tuberculous; although grave suspicion of an acute pulmonary tuberculosis may have existed and apparently be confirmed by the tuberculin test. Much needless alarm may therefore be produced by a positive reaction, which really demonstrates only that somewhere the child has a tuberculous focus, but it does not prove that his present disease is of a tuberculous nature.

Shortly before death, whether from general or any form of localised tuberculosis, as a rule there is no reaction to any of the local tests. Likewise, a child in an extremely asthenic condition from any cause whatever may give no reaction although he has a latent tuberculosis. During active measles also the test is of little value. No conclusions therefore can be drawn from tests made under these conditions. On the whole von Pirquet's cutaneous test is to be preferred for general use.

**Tuberculides of the Skin.**—These are at times of considerable value in the diagnosis of tuberculosis in general.<sup>1</sup> Although seldom seen in the acute varieties, they are not uncommon in the more slowly progressing forms. The distribution of the lesions is fairly constant. They are found chiefly on the buttocks, lower abdomen, genitalia and thighs. The number present is generally small, half a dozen to a dozen; but they are sometimes numerous and may be widely distributed. The lesion begins as a minute red papule, which is soon surmounted by a small vesicle. This dries to form a crust. If the crust is removed a small pit-like depression remains, which heals quickly, leaving a white, glistening scar surrounded by a pigmented border. The lesion runs its entire course in two or three weeks. Tubercle bacilli are often present but difficult to demonstrate. Tuberculides of the skin in young children are evidence of a widely disseminated process and are a very bad prognostic sign. Such patients rarely survive more than a few weeks.

**General Prognosis of Tuberculosis.**—The outlook for a young child with general or pulmonary tuberculosis is always bad. So long as the disease remains confined to the lymph nodes, the child is not usually in

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<sup>1</sup> Tileston, Archives of Internal Medicine, July, 1909.

danger, except from accidents connected with their softening and ulceration, which after all are rare. Spontaneous cure may occur in these glands in the same way as in others in the body, viz., by encapsulation, calcification, etc. Such a result is no doubt a very frequent one; exactly how often it occurs it is impossible to say. But when once the disease has gained any headway in the lung itself, its steady advance is almost certain in a young child. In those who are older and have more resistance the chances of an arrest of the process are much greater.

If the bacilli have gained entrance into the body in any considerable numbers, even though they are shut up in an encapsulated, caseous, bronchial gland, the patient is never free from the danger of general infection.

**Prophylaxis.**—The prevention of tuberculosis must have constant reference to its cause. The first essential is the destruction of the tubercle bacilli wherever they exist. Since most of those existing in the air are derived from the sputum of patients affected with pulmonary tuberculosis, it should be insisted upon, everywhere and at all times, that the sputum from such cases should be collected in special cups or cloths and destroyed either by germicides or by fire. The next point is to avoid needless exposure. A tuberculous mother should on no account nurse her child nor kiss it upon the mouth. A wet-nurse likewise should be free from any tuberculous taint. No nurse or other care-taker should ever be employed about children who has, or ever has had, pulmonary tuberculosis. It is wise to exclude also those who suffered when children from tuberculosis of the bones or the cervical glands, although the danger from such persons is extremely slight. If active tuberculosis exists in any member of the family, a young child should be kept away from the room, and if possible should not reside in the house. On no account should infected persons be allowed to kiss children or sleep in the same bed with them. The danger from drinking-cups and other dishes should not be forgotten. A tuberculous person should either have his special dishes, or the utmost care should be taken to boil all those which he has used. Cows whose milk is used for children should be under regular veterinary inspection and should have passed the tuberculin test. In any case when the slightest doubt regarding the health of the cows exists, or when the source of the milk is unknown, the milk should be heated to a temperature of 140° F. for forty minutes. The danger of infection through the alimentary canal is very much less than through the respiratory tract, and consequently the precautions first mentioned are much more important than those relating to the food, although the latter should on no account be neglected.

In the case of delicate children and those with tuberculous parents or with other tuberculous near relatives, everything possible should be done to fortify them against the disease. They should be kept under more or

less constant medical supervision. Attacks of bronchitis or bronchopneumonia should be watched with the greatest solicitude. Exposure to influenza, measles or pertussis should especially be avoided. The country rather than the city should be chosen for residence, and the child should spend the winter and spring in some warm, dry climate. Parents should be distinctly taught that watchfulness and care do not mean coddling or the keeping of children in the house the greater part of the time. Such children should live as much as possible in the open air, and every form of sport encouraged which tends to keep them there. Overheated houses are one of the most prolific agencies in perpetuating a delicate condition of health. Plenty of fresh air in sleeping apartments should always be insisted upon. All catarrhal troubles of the nose and pharynx should receive early and prompt attention, especially should hypertrophied tonsils and adenoid growths of the pharynx be removed, since these are conditions which form a most favourable nidus for the growth of tubercle bacilli.

**Treatment of General and Pulmonary Tuberculosis.**—If fresh air and a proper climate are necessary for the cure of this disease in adults, they are tenfold more necessary in the case of children. Without them there is little hope for a child with active pulmonary tuberculosis. Nowhere do these cases do so badly as in a hospital located in a city, and no class of hospital cases do worse than these. The same regions that are beneficial for adult cases usually agree with children, with the exception that the latter, as a rule, do better in a warm than in a cold climate. Plenty of fresh air and sunshine are essential. A child must be where he can be kept in the open air for the greater part of each day, in spite of fever, cough, or other acute symptoms.

For the most acute cases when the children are confined to the bed, the largest, best-ventilated, and sunniest room available should be secured, and the windows should be constantly open. The general management of such cases is the same as for those with acute pneumonia.

No specific remedy for tuberculosis has as yet stood the test of experience. The diet is a matter of the utmost importance. Tuberculous patients must be fed like most other sick children, care being taken not to disturb the digestion by the unnecessary use of drugs. For a staple article of diet, milk is the best, and when this is not well borne some of its substitutes—buttermilk, kumyss, matzoon, etc.—may be tried. Cream is almost as useful as cod-liver oil, and should be given in one form or another whenever the child's digestion can tolerate it.

Tuberculin in the treatment of this disease in young children has as yet been too little employed to enable one to form any definite conclusions as to its value. Its application should be directed by the same rules as those employed in adults. It is a therapeutic procedure which deserves more attention than it has hitherto received.

The two drugs which are most useful are creosote and cod-liver oil. Creosote may be given both by the stomach and by inhalation, as in cases of pneumonia. By the stomach there may be used for older children, the shellac-coated pills or capsules containing one or two drops of creosote; it may be given in conjunction with cod-liver oil. Cod-liver oil is usually best given in a fresh emulsion, although some children bear the pure oil better than its preparations. Inunctions of this or other oils are of some value when not well tolerated by the stomach. Arsenic, iron, and the compound syrup of the hypophosphites are all useful as general tonics, but as specifics their action is very questionable.

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## CHAPTER XI.

### SYPHILIS.

SYPHILIS is a communicable disease due to a specific organism, the *spirochæta pallida* of Schaudinn. In acquired syphilis this is found in the primary lesion, in the mucous patches and in the lymph nodes. In hereditary syphilis it is found in the cutaneous lesions, in the fissures at the angle of the mouth and in the mucous patches of the buccal cavity, with less regularity, in the internal organs, especially the liver and spleen. While in the still-born child and in early cases, the number of organisms found is very great, they are not so numerous at a later period, and they diminish rapidly after treatment is begun. In the late lesions the spirochætæ are not numerous, and are difficult to demonstrate.

In infancy and childhood both the acquired and the hereditary forms of syphilis are seen.

### ACQUIRED SYPHILIS.

While acquired syphilis is very much less frequent than the hereditary variety, it is by no means a rare disease in early life. It is not improbable that some of the manifestations of syphilis in later childhood which are usually denominated "late hereditary syphilis," are really due to the acquired form.

**Etiology.**—An infant may be infected by its mother during parturition; but this is extremely rare and can take place only when there are lesions upon the mother's genitals. Infection is more likely to be from a mother who contracts syphilis subsequent to the birth of the child, and may occur through nursing or accidental contact by kissing, etc. In either of these ways children may be infected by wet-nurses, or from a venereal sore upon the nipple. Whether syphilis can be communicated



through the milk when the nipple is perfectly healthy and free from fissures, is somewhat doubtful.

Syphilis may be communicated directly from a syphilitic child to one who is healthy by kissing, by sexual contact, or indirectly by means of bottles, spoons, cups, clothing, etc. The latter mode of infection is most likely to occur in institutions. Vaccination was formerly a not infrequent mode of communicating syphilis, but since the general introduction of bovine virus this is very rarely seen. Cases have been recorded where the disease has been conveyed by the rite of circumcision, either from the mouth or the instruments of the operator.

The relative frequency of the different sources of infection is shown by Fournier's statistics of 40 cases: The source of infection was the parents in 19; nurses, in 8; servants, in 4; sexual contact, in 4; vaccination, in 2; other children, in 2; a physician, in 1. The ages at which the disease was acquired in this series of cases were as follows: During the first year, 19; during the second year, 10; during the third and fourth years, 7; from the fifth to the fourteenth year, 6.

**Symptoms.**—The symptoms of acquired syphilis in children are in all respects similar to the same disease in the adult. A primary sore is present at the site of infection, which is most frequently the lips, the mouth, or some part of the face; very rarely is it seen on the genitals. There are very few individual symptoms belonging to hereditary syphilis which may not also be present when the disease is acquired. Its course, however, is very much milder in the latter and a fatal termination is rare. Fournier states that of his forty-two cases only one died of marasmus. This marked contrast to hereditary syphilis is due chiefly to the fact that in the acquired variety the infant is rarely affected during the early months of life, a time when hereditary syphilis is so very fatal.

Tertiary symptoms may appear at any time from three to twenty years after the original infection.

The treatment is the same as that of hereditary syphilis.

#### HEREDITARY SYPHILIS.

**Etiology.**—If both parents are syphilitic, the child is usually but not invariably so. The symptoms, however, are not more severe than when the inheritance is from one parent only. The likelihood of transmission depends upon the stage of the disease in the parents. If the mother is suffering from secondary symptoms, transmission is almost certain. If active treatment has been employed for several months, if the child is born at a period when no active symptoms are present, or if the symptoms are of a tertiary character, the offspring will probably escape. First-born children are more likely to suffer severely from syphilis than the later ones, provided infection of the parents has taken place prior to the birth of all the children.

The transmission of syphilis from the father without the intermediate infection of the mother was once held to be possible. At the present time, however, this question must be placed among those not yet definitely settled. There can be no doubt that in the vast majority of the cases the infection of the child is from the mother.

If both parents are healthy at the time of conception and the mother becomes infected during her pregnancy the child may or may not be syphilitic. Transmission to the child is much less likely to occur if the mother is infected during the last two months of her pregnancy than earlier, although, as Hutchinson's cases conclusively show, there is no certainty that the child will escape. Diday states that if the mother is infected before the fourth week and proper treatment is instituted, the child will usually escape on account of the relation of the embryo to the maternal circulation during this early period.

In 1837 Colles enunciated the following proposition, the truth of which has been abundantly verified since his time: "A new-born child affected with inherited syphilis, even although it may have symptoms in the mouth, never causes ulceration of the breasts which it sucks if it be the mother who suckles it, although continuing capable of infecting a strange nurse."

Caspary inoculated with syphilis a woman, apparently healthy, who had aborted with a syphilitic child; the result was negative. A similar experiment was made by Neumann, with a like result. Widal reports a case of an apparently healthy woman who had a syphilitic child by an infected husband; later, by a second husband who was free from syphilis, she had a syphilitic child. The conclusion seems irresistible that the carrying of a syphilitic child gives immunity to the mother against the disease, and that this immunity is due to the fact that she herself suffers from syphilis, or a modification of that disease. The mother under these circumstances can not be inoculated, either by her syphilitic nursing infant or artificially.

That hereditary syphilis is contagious is conclusively shown by a number of recorded instances in which a healthy wet-nurse has been infected by a syphilitic infant. However, such examples of contagion are very rare, and many writers of large experience state that they have never seen it. It is certainly true that the danger of spreading infection from a case of hereditary syphilis has been exaggerated.

**Lesions.**—Death may be due to syphilis, and yet the autopsy may reveal no characteristic anatomical changes, and in fact there may be no demonstrable changes in any of the organs except the presence of the spirochæta.

**Bones.**—In the case of a syphilitic fœtus, a still-born child, or one dying soon after birth, the changes in the bones are more uniformly present than are any other lesions. They are, in fact, rarely wanting,

and it is by them usually that syphilis is recognised post mortem. The long bones are principally affected, the most important changes being found at the junction of the shaft with the epiphyseal cartilage. The lesion is termed an epiphyseal osteo-chondritis or acute epiphysitis. There is in the early stage congestion, swelling, and cell proliferation, which may be followed by separation of the epiphysis, suppuration in the neighbouring joint, osteomyelitis, and necrosis. These changes are more fully considered under Diseases of the Bones.

*Liver.*—This is probably more frequently involved in the foetus and newly-born infant than any other organ. The syphilitic lesions of the liver consist in an interstitial hepatitis, a gummatous hepatitis, or a combination of the two varieties.

In the interstitial form, which is most frequent in infancy, there is first a congestion and swelling of the organ, with the exudation of leucocytes in groups. The liver is enlarged, frequently very much so, but presents few other gross changes. Later, new connective tissue forms, and atrophy of the liver cells takes place, with obliteration of some of the portal and hepatic vessels. This process may be diffuse, but it is usually in patches. Groups of miliary syphilomata may also be found. If the process is diffuse, the liver is large, firm, and of a grayish-yellow colour. If it is localised, the affected areas are yellow or gray and the other parts are normal.

The gummatous form is not frequent in early infancy, but belongs to a little later period. In this there may be miliary syphilomata with interstitial changes, and in addition the formation of small or large gummatous tumours, which may be softened at the centre. They are surrounded by zones of new connective tissue and the liver cells are atrophied. Amyloid changes may be present.

In the late form of hereditary syphilis, usually seen in children over four or five years old, the liver is occasionally affected. The lesions resemble those of the congenital variety. There are found cirrhotic changes, which may be diffuse or circumscribed, and gummatous deposits, which vary from a minute size to that of a cherry; there may be amyloid degeneration.

*Spleen.*—This is almost invariably enlarged in newly-born children with syphilis and in syphilitic foetuses, but nothing characteristic is found under the microscope. In older children the enlargement of the spleen is apt to be greater; the organ may be the seat of interstitial changes, and sometimes there may be gummatous deposits. These changes are rare in children under two years of age.

*Respiratory System.*—In syphilitic infants who are still-born and in those who die soon after birth, there is frequently found in the lungs what is known as "white pneumonia." This process consists in fatty changes in the epithelium of the air vesicles; with this there is associated

a certain amount of interstitial pneumonia, which is chiefly peribronchial. In older cases the interstitial pneumonia is extensive, and the lungs may be the seat of gummatous deposits, which soften and form small cavities. Accompanying these changes there may be bronchiectasis, emphysema, and the usual secondary lesions which follow chronic interstitial pneumonia. In syphilitic infants there is a strong tendency for all inflammations of the lungs to become chronic.

The trachea and bronchi are in rare cases the seat of stenosis, which results from cicatrization following the softening of gummatous deposits in their walls. Lesions of the larynx are also infrequent. There is usually perichondritis, which more often involves the epiglottis than any other part, and sometimes there is the formation of papillomatous masses; but ulceration and stenosis are both rare.

The nasal mucous membrane in the early stage of the disease is very constantly the seat of a chronic catarrhal inflammation, which may be accompanied by superficial ulceration. In the late cases there is deeper ulceration, from the breaking down of gummata, with extension to the periosteum, cartilages, and bones, causing perforation of the septum, necrosis of the bones, etc.

*Nervous System.*—Syphilitic lesions of the brain and cord are rare in children as compared with adults, and they are especially so in infancy. The most characteristic cerebral lesion of the newly-born child is hydrocephalus, which may depend upon ependymitis, as in two cases reported by D'Astros, the disease proving fatal in the second month. Syphilitic meningitis is exceedingly rare under two years. There is occasionally seen in young infants a chronic basilar meningitis of syphilitic origin. Chronic pachymeningitis associated with gummata has been observed as early as the fourth year. There have been reported in infants a few cases of chronic meningitis with great thickening of the dura mater and cerebral sclerosis.

Nearly all the syphilitic lesions of the nervous system which are seen in adult life have been observed in childhood, but infrequently, and in young children they are extremely rare.

*Heart and Arteries.*—These may be affected even in young infants. Adler, of four cases examined, found two in which well-marked lesions were present in infants under four months. There was endarteritis of the coronary arteries accompanied by the early changes belonging to interstitial myocarditis. Chiari has reported syphilitic endarteritis of the brain at fifteen months, followed by thrombosis and softening.

*Digestive System.*—Chronic catarrhal pharyngitis is almost a constant symptom of the early cases. Later there is seen superficial or deep ulceration of the pharynx, tonsils, or fauces, which may lead to perforation of the soft palate or to the formation of condylomata.

There are no important lesions of the stomach or intestines either

with early or late syphilis. The rectum is occasionally the seat of ulceration, and condylomata may form even in young children.

*Organs of Special Sense.*—Otitis is a frequent accompaniment of the early syphilitic pharyngitis. It is very likely to become chronic, and in many cases results in a permanent impairment of hearing. Iritis is relatively rare in children, but it may occur even in intra-uterine life, as shown by the presence of adhesions in newly-born children. It is usually seen in infants four or five months old, and is always serious. Interstitial keratitis occurs frequently as a late manifestation of syphilis. Choroiditis and optic neuritis are both occasionally seen, but they are rare.

*Genito-urinary Organs.*—Nearly all these may be affected, but generally in the late period of the disease. There may be chronic interstitial nephritis and more rarely gummatous deposits in the kidney, interstitial changes in the suprarenal bodies, and orchitis, which usually affects the body of the organ, rarely the epididymis; it is generally an interstitial inflammation, with or without gummatous deposits.

Among the less frequent visceral lesions may be mentioned abscesses of the thymus, which are usually small and multiple; enlargement of the pancreas, with an increase of connective tissue and glandular atrophy; and chronic peritonitis. The lesions of the mucous membranes will be considered under Symptoms.

*Symptoms.*—As the result of syphilis, abortion may take place at any period of pregnancy, with the discharge of a dead or macerated fœtus, or the child may be still-born at term, or it may be born alive prematurely, but with so feeble a vitality that it survives but a few hours. Under these circumstances it is often difficult and sometimes impossible to decide positively with reference to the existence of syphilis. Maceration of the fœtus or peeling of the skin is no proof, and even the examination of the internal organs may not be conclusive, except for the presence of spirochætæ. Lomer examined 43 fœtuses, all dying before the thirtieth week of pregnancy; he found the spleen and liver enlarged in all, and marked bone changes in 21. Birch-Hirschfeld examined 108 newly-born syphilitic infants; he found the spleen invariably enlarged; typical bone changes were present in 35, but in many cases the bones were normal. Mervis, from an examination of 92 syphilitic fœtuses, states that no eruption upon the skin was found earlier than the eighth month.

Symptoms are present at birth in only a small number of cases. In such there is usually a very severe degree of infection, and the infants do not often live more than a few days. Upon the skin there may be seen an eruption of pustules, papules, or bullæ. The bullæ are usually upon the soles and palms, but may be found upon other parts of the body. The name "syphilitic pemphigus" is often given to this condition. The bullæ are at first small, and then coalesce and form larger ones two inches or more in diameter. They contain a turbid serum which is sometimes

tinged with blood, and sometimes yellow from pus. Pustules, when present, are usually seen upon the face or scalp. The general appearance of these infants is wretched in the extreme. The body is wasted, the skin wrinkled, and temperature subnormal. The spleen is usually enlarged and often the liver also. Death usually occurs from inanition within two weeks.

In the great majority of cases the infant appears healthy at birth, and continues so for a variable time before the manifestation of the characteristic symptoms of syphilis. As a rule, the more intense the infection, the earlier the symptoms make their appearance. The earliest symptoms are generally seen between the second and the sixth weeks. If three months pass without evidence of syphilis, the probabilities are that the child will escape. Miller (Moscow) gives the following statistics of the time of beginning of symptoms in 1,000 cases:

|   |           |
|---|-----------|
| Symptoms appeared during the first week | 85 cases. |
| “ “ “ “ second week                     | 138 “     |
| “ “ “ “ third week                      | 240 “     |
| “ “ “ “ fourth week                     | 177 “     |
| “ “ “ “ fifth week                      | 86 “      |
| “ “ “ “ sixth week                      | 54 “      |
| “ “ “ “ seventh week                    | 50 “      |
| “ “ “ “ eighth week                     | 30 “      |
| After the eighth week                   | 140 “     |

Sometimes the constitutional symptoms—wasting, cachexia, etc.—are noticed before the local ones, but usually this is not the case. Generally the first symptom is the coryza or “snuffles,” which resembles an ordinary cold in the head, except that it persists. It is accompanied by a hoarse cry, indicating that the larynx participates in the catarrhal inflammation. Soon the eruption makes its appearance, being generally first seen upon the hands, feet, and face. Fissures and mucous patches may be seen upon the lips, about the anus, and elsewhere. There is often slight fever, from 99° to 101° F. There may also be observed excessive tenderness and swelling about the shoulders, elbows, wrists, or ankles, due to acute epiphysitis, which may cause the child to cry from the slightest amount of handling, and the limbs may be moved so little that paralysis is suspected.

In a severe case, as these local symptoms develop, the infant's general nutrition suffers. He loses steadily in weight, he becomes extremely anæmic, and whines and frets almost continually, but especially at night. The features have a pitiful, drawn expression; and the face is wrinkled, giving the infant a very old appearance. The skin has a peculiar sallow colour, which has been well described as *café au lait*. The symptoms may continue until a condition of extreme marasmus is reached, or death occurs from some intercurrent affection of the lungs or digestive organs.

In the milder forms of infection the severe constitutional symptoms described are not seen, although the local evidences of disease are well marked. The severity of the symptoms is also much modified by treatment, especially when this is begun early.

The most important local symptoms are the coryza, eruption, fissures about the mouth and anus, mucous patches, painful swellings at the extremities of the long bones, pseudo-paralysis, and onychia.

*Coryza*.—In most of the cases this is the first symptom. Beginning like an ordinary catarrh, it is distinguished by its severity and its persistence. There is a copious discharge of mucus and serum, often tinged with blood. Thick crusts form, which produce the usual symptoms of nasal obstruction; there is great difficulty in nursing; the infant breathes through the mouth, and the mucous membrane of the mouth is dry, causing great discomfort. If untreated, the process, which at first involves the mucous membrane only, may extend to the submucous tissue, causing ulceration; but the cartilages and the bones of the nasal fossæ are not involved till a later period in the disease.

The nasal catarrh is associated with more or less laryngitis, causing hoarseness or aphonia, and rarely there may be laryngeal stenosis. Dillon Brown has reported one case in an infant six weeks old, which recovered after intubation.

*Eruption*.—The early eruption usually appears after the coryza has lasted about a week; but the two may come at the same time; or the coryza may be absent or so slight that the rash seems to be the first symptom.

Occasionally there is seen a diffuse blush or roseola, but more frequently the eruption is macular, occurring in small, dark-red spots about the size of the infant's finger nails, usually circular and often slightly elevated; there is no surrounding inflammation, and rarely any itching. It is usually most abundant upon the face, the neck, and the extensor surface of the upper and lower extremities, especially the hands and feet, sometimes extending over the entire body, although it is generally scanty over the chest and abdomen. At first the



FIG. 210.—EARLY ERUPTION OF HEREDITARY SYPHILIS. Infant two months old.

colour is bright, but gradually becomes of a dusky-red or coppery hue. After a little time very fine scales may be seen upon the surface of the red macules. The rash comes out slowly, usually requiring from one to three weeks for its full development. It fades gradually, leaving a coppery discolouration of the skin, which continues for a long time. The duration of the eruption is from three to eight weeks; less if active treatment is employed.

A papular eruption is rarely seen alone, but is usually associated with the macular variety. The papules are of a brownish colour and are hard. They are seen most frequently upon the palms and soles.



FIG. 211.—EARLY ERUPTION OF HEREDITARY SYPHILIS. Infant two and one-half months old.



FIG. 212.—SYPHILITIC SCALING OF THE FOOT. From an infant eight weeks old.

A squamous eruption is frequently seen upon the palms and soles, but very rarely elsewhere. In a few cases this scaliness forms the most distinctive feature of the cutaneous lesion (see Fig. 212).

*Fissures and Mucous Patches.*—These are among the most diagnostic features of early hereditary syphilis. Fissures are most frequently seen on the lips and about the anus, but they may occur about the nostrils and occasionally elsewhere. The fissures of the lips are really linear ulcers, and are distinguished by their persistence in spite of local treatment. They are multiple, deep, painful, and bleed easily. After healing, these fissures may leave many cicatrices radiating from the mouth, the contraction of which produces the so-called “purse-string deformity.”

Mucous patches may develop from fissures, but more frequently from papules which are situated in regions where they are exposed to constant



moisture and friction. They are very common upon the muco-cutaneous surfaces and wherever the skin is especially thin. They are most apt to be seen about the lips, anus, scrotum, and vulva, but they may also be found behind the ears, between the toes, in the folds of the groin, axillæ, or buttocks. They vary from an eighth to half an inch in diameter, are whitish in colour, and are raised rather than excavated.



FIG. 213.—A LATER FORM OF ERUPTION IN HEREDITARY SYPHILIS.  
Infant eight months old.

Ulcers may be present upon any of the mucous membranes, frequently in the mouth or on the genitals: they are seldom symmetrical, and while they may be broad they are never deep.

*Hæmorrhages.*—They are generally associated with the lesions of the mucous membranes, especially of the nose. In young infants with severe infection, bleeding may occur from the bullous eruption upon the skin, or from the fissures at any of the orifices, particularly the mouth and anus. Fischl has reported seven cases of multiple hæmorrhages in the newly born, associated with other symptoms of congenital syphilis. Mracek noted hæmorrhages in thirty-three per cent of 160 autopsies on syphilitic still-born infants or those dying soon after birth. Examination of the blood-vessels in some of these cases showed infiltration of their walls and narrowing of their lumen. The vascular changes were thought to be the cause of the bleeding.

*Nails.*—The nails present several peculiarities in syphilitic infants. There may be a disease of the matrix resulting in suppuration and exfoliation of the nail: frequently the dorsum is much arched, and the nail appears as if it had been pinched by a pair of forceps—i. e., claw-shaped; this is an early symptom of some diagnostic importance. The hair and eyebrows frequently fall out completely. This symptom is not usually present in very early infancy.

*Pseudo-paralysis.*—This is due to acute epiphysitis, and it may be the first symptom of hereditary syphilis to attract attention. It is usu-

ally noticed when the infant is a few weeks old that one or sometimes both arms are not moved, and that the parts are tender when handled. The arm is very frequently held in marked inward rotation with the palm looking outward, resembling the position in Erb's palsy; but careful examination makes it evident that the loss of power is only apparent, and that it is due either to the pain which motion produces or to epiphyseal separation. A history will usually be obtained that loss of power did not exist at birth, but developed subsequently. The electrical reactions in these cases are normal, and the rapid improvement under mercurial treatment is diagnostic.

The only visceral symptoms of importance are, enlargement of the spleen, which is almost invariably present in the active stage of hereditary syphilis, and jaundice with or without enlargement of the liver.

**Late Hereditary Syphilis.**—The symptoms may come on at any period during childhood or about the time of puberty, but very rarely at a later time than this. They are seen both in those who have had the usual symptoms of hereditary syphilis in early infancy, and in others where the most careful examination into the history fails to disclose any symptoms whatever of early syphilis. It is fair to assume in such cases either that early symptoms were absent or that they were of trivial importance.

Late hereditary syphilis shows itself by symptoms which in acquired disease would be classed as tertiary. The most characteristic are the affections of the teeth, the bones, gummatous deposits in the solid viscera, the skin, or mucous membranes, the breaking down of which may lead to ulceration.

**Teeth.**—There are no peculiarities in the first teeth of syphilitic children except their proneness to early decay. They are rather more likely to appear early than late.

The characteristic teeth of syphilis are those of the second set. In estimating the diagnostic value of these changes, only the upper central incisors are to be relied upon; these are the test teeth. Although changes are frequently seen in other teeth, they are not always diagnostic. Typical syphilitic teeth, according to Hutchinson, have each a single notch in the centre of the edge (Fig. 214). The notch is usually shallow and more or less crescentic in shape. The enamel is generally deficient in the centre of the notch, and the tooth here is apt to be discoloured. The teeth in other cases are variously dwarfed and deformed. (See Fig. 215.) They often taper regularly from the base to the edge, giving rise to the term "screw-driver teeth." The teeth are not so flat as the normal



FIG. 214.—TYPICAL "HUTCHINSON'S TEETH." (After Fournier.)

incisors, but often rounded and peg-like. They are not properly placed, but incline either toward or away from each other. They are seldom large enough to touch the adjacent teeth on both sides.

Although Hutchinson's teeth may generally be taken as conclusive evidence of syphilis, they are not invariably so, as Keyes and others have shown. It is to be remembered in this connection that the absence of changes in the teeth is of no importance whatever as evidence that syphilis is not present. Hutchinson states that they are wanting in more than half the cases.

*Bones.*—The form of disease which is usually seen at this period is an osteo-periostitis, affecting principally the shaft of the long bones and the cranium. It has already been described elsewhere.

*Lymph Nodes.*—They are less frequently affected than in adults, and in early infancy they are seldom much involved. In most cases after the first year there may be found a moderate degree of enlargement of the post-cervical and epitrochlear glands, swelling of the latter having considerable diagnostic value. Under normal conditions the latter can scarcely be felt; but in syphilitic children they may be as large as a pea or a small bean; sometimes two or three of them can be distinguished. They are so rarely enlarged from other constitutional conditions that, provided no local cause for the swelling exists, they should always create a suspicion of syphilis. The post-cervical glands are frequently affected, but are not so diagnostic. The degree of enlargement is rarely great. Occasionally there are seen in the neck large masses of swollen lymph glands which resemble tuberculous swellings. They are, however, very rare.

*Special Senses.*—The most frequent affection of the eye in late syphilis is interstitial keratitis, the close connection of which with hereditary syphilis was first pointed out by Hutchinson. It is usually found associated with the typical notched teeth. The diagnostic value of keratitis in syphilis is denied by Fournier, who states that, while often syphilitic, it is not infrequently due simply to malnutrition. Both eyes are usually affected, and in all degrees of severity, from a slight haziness of the cornea to complete opacity. However, with an early diagnosis and prompt treatment, a marked degree of improvement may be expected in most cases.



FIG. 215.—SYPHILITIC TEETH. Boy eight years old; under observation several years with various syphilitic manifestations.

Chronic otitis may be a result of the acute process seen in early infancy. There is nothing peculiar about the inflammation in these cases. A form of deafness occurs in older children, which Hutchinson states is almost invariably due to syphilis. Its onset is quite sudden, without pain. The loss of hearing is apt to be permanent, and if it occurs early in childhood it is a cause of deaf-mutism.

*Skin.*—The most important of the later manifestations of syphilis consists in the formation of subcutaneous gummata. In the early stage they are indurated, elastic, of a grayish colour, with red borders. Under treatment they disappear quite rapidly by absorption; but when neglected they break down, leaving large deep ulcers. These ulcers are quite characteristic in appearance, but may be confounded with those due to tuberculosis. The syphilitic ulcer has rounded, thickened, indurated borders, and a base which is depressed and has the appearance of being scooped out. It is sometimes covered by hard crusts and is surrounded by a red areola. It leaves a smooth white scar. The most frequent situation is upon the face and upper part of the legs or thighs. Tuberculous ulcers have usually soft, flat edges, and do not extend so deeply; they are more irregular in outline; the cicatrix left is of a purplish colour, which becomes red and slowly fades. Tubercle bacilli may be found.

*Nose and Palate.*—Disease of these parts generally begins as the breaking down of gummatous deposits in the mucous membrane. The nose may in consequence be the seat of a protracted fœtid discharge (ozæna). The disease may take on a destructive form of ulceration which is at times phagedenic, and may cause rapid destruction of the nasal cartilages and bones, perforation of the septum, and occasionally of the floor of the nasal fossæ. There may be necrosis of the turbinated bones, the vomer, or the ethmoid. In the most severe forms the nose may be almost destroyed in the course of a few weeks. There may be at the same time deep ulceration of the soft palate, leading to perforation. In a young person this is almost invariably due to syphilis. In many particulars these ulcerations of the nose and palate resemble lupus; they are distinguished by the rapidity of their progress, syphilis often doing as much damage in weeks as is done by lupus in years.

*Other Symptoms.*—Syphilitic disease of the larynx and bronchi is rare in childhood. The former may give rise to hoarseness or aphonia and occasionally to stenosis; the latter to a chronic cough and asthmatic attacks. There are no characteristic symptoms belonging to syphilis of the lungs. The different lesions of the central nervous system which may be due to syphilis are all quite rare. The forms have already been mentioned, and their symptomatology is discussed in Diseases of the Nervous System.

The only visceral changes which aid much in diagnosis are those of

the liver and spleen. The liver is often enlarged, sometimes to a marked degree, and occasionally there is ascites, but very seldom jaundice.

Enlargement of the spleen is a very frequent symptom—in fact, it is almost constant during active syphilitic disease. I have several times seen it so swollen as to form an abdominal tumour of considerable size. In one case, in a boy three years old, the spleen extended five inches below the free border of the ribs, quite to the crest of the ileum. It was associated with moderate enlargement of the liver, as is usually the case.

In addition to the local symptoms of late hereditary syphilis enumerated, there are others of a general character which are quite as important. The body is usually undersized; the constitution is delicate, and shows but little resistance to all forms of disease; puberty is frequently delayed, and the development of the breasts and the genital organs often imperfect; anæmia is usually present, and the skin has a sallow appearance. Mentally, many of these children are somewhat deficient, and in a few instances they become idiotic, epileptic, or the subjects of dementia.

**Diagnosis.**—The diagnosis of early syphilis in most cases is not difficult. The coryza, eruption, labial fissures, mucous patches about the anus and genitals, enlarged spleen, and later the general cachexia—all unite to form a picture which it is difficult to mistake. In irregular cases the diagnosis is easy just in proportion to the number of the foregoing symptoms which are present. Special care should be taken not to confound the moist papules of simple intertrigo upon the buttocks or thighs with those of syphilis. In doubtful cases much assistance may be obtained from the discovery of the spirochætæ in the external lesions and from the Wassermann reaction.

In late syphilis the following symptoms are the most reliable for diagnosis: notching of the teeth, falling in of the bridge of the nose, interstitial keratitis, deafness not traceable to ordinary otitis, enlargement of the spleen and epitrochlear glands, ulceration of the palate or nose, the sabre-like deformity of the tibia, and nodes upon the tibia or cranium. There are often found in older children indefinite symptoms in regard to which a suspicion of syphilis exists. For such cases the Wassermann test is of very great value.

It becomes at times important to distinguish hereditary from acquired syphilis. Visceral lesions in acquired syphilis are not common and belong to the late period of the disease; in the hereditary form they are well-nigh constant and occur early, often being present at birth. The acute epiphysitis, sometimes accompanied by pseudo-paralysis, seldom if ever occurs in acquired syphilis, though frequent in the hereditary form. Symptoms due to defects in development, like the misshapen finger-nails, are seen only in hereditary syphilis. The early symptoms re-

ferable to the mucous membranes and mucocutaneous surfaces—coryza, hoarseness, hæmorrhages, labial fissures, etc.—so characteristic of hereditary syphilis, have no place in the acquired form, while the single primary lesion sometimes found in the acquired form does not exist in the hereditary disease.

**Prognosis.**—Generally speaking, the prognosis is worse in infantile syphilis than in that of adults. In infancy it is much worse when hereditary than when acquired, for the reason that often the child who is the subject of hereditary syphilis has been affected by the poison from the very beginning of its existence, and this has modified its entire development.

The results of 206 syphilitic pregnancies observed by Jullien (Paris) were as follows: Abortion occurred in 36, stillbirths in 8, and 69 children died soon after birth, making a total mortality of 55 per cent; 50 were living and syphilitic; only 43 living and in good health. Still worse were the results in cases observed by Le Pileur: Of 154 pregnancies in syphilitic women, there were 120 abortions or stillbirths, 26 children died soon after birth, and only 8 survived. The statistics of the Foundling Asylum in Moscow for ten years showed that of 2,038 syphilitic infants the mortality was over 70 per cent.

Such a mortality as that indicated in the above statistics is seen only in institutions where little or no previous treatment has been employed. In private practice certainly nothing approaching it occurs.

In addition to those who die early as the result of syphilitic infection, there must be added many whose constitutions are so impaired by syphilis that they fall an easy prey in infancy to pneumonia, diarrhœa, or other forms of acute disease. The remote effects of syphilis in infancy it is hard to estimate; it may exert an injurious influence upon the constitution in childhood and even throughout the life of the individual.

The prognosis in an individual case depends upon the age at which the symptoms develop, the time when treatment is begun, upon its thoroughness, and upon the surroundings and mode of nourishment of the child. The outlook is better the longer after birth the first symptoms appear; it is also better in infants who are nursed than in those who are artificially fed.

As compared with syphilis of the adult, relapses are rare, and when they occur early they are nearly always the result of insufficient treatment. If proper early treatment is carried out, the severe late symptoms are rare; patients are usually free from all symptoms until six or seven years old, or until near the time of puberty—two periods when they are likely to develop.

The prognosis is better in the later children of syphilitic parents than in the earlier ones, provided infection has preceded the birth of all the children. This fact illustrates the general tendency of the syphilitic

poison to diminish in virulence as time passes, even without treatment. The following instance cited by Bertin well illustrates this point:

In the first pregnancy, the mother aborted with a dead child at the sixth month; in the second, at the seventh month; in the third, at seven and a half months; in the fourth the child was born at term, and lived eighteen days; in the fifth it lived six weeks; in the sixth the child lived four months, without treatment.

**Prophylaxis.**—No infected person should be allowed to marry until at least two years have passed after the initial sore, treatment being continued meanwhile; nor if there are any active symptoms, no matter how long a time has elapsed since infection. There is no certainty in any case that the child will escape.

The mother should be treated during her pregnancy: (1) If she is syphilitic, whether the disease was acquired at the time of conception or subsequently; (2) if the father is known to be suffering from syphilis, whether the mother has symptoms or not; (3) if the mother has ever previously shown signs of syphilis, even if she has had no active symptoms for a considerable period. In all these conditions if efficient treatment is carried on throughout pregnancy there is a strong probability, but in no case a certainty, that the child will escape. The third condition mentioned is the one in which treatment is most likely to be neglected, especially if the mother has previously borne a child who was not syphilitic. Syphilis, however, shows a strong tendency to reappear and become active during pregnancy, even though it has been long quiescent, as the following case cited by Diday shows:

A woman who had lost seven children from syphilis was put under treatment during the eighth pregnancy; result—child born healthy, and continued so. In the ninth pregnancy treatment was continued with a like result; in the tenth pregnancy, no treatment, child syphilitic, dying when six months old; in the eleventh pregnancy, treatment repeated, child healthy.

The danger of infection during labour is slight. As the greatest danger of infecting a child after birth is from its parents or a wet-nurse, syphilitic parents should be duly warned of the danger to their children, and especially should be cautioned against kissing them or sleeping in the same bed with them. The utmost care should be exercised to prevent a healthy child from being infected by a syphilitic nurse. A nurse should never be accepted without a thorough examination, no matter how clear a history may be given. As a syphilitic child in the household may be the means of infecting other children, the same precautions should be taken as in the case of other contagious diseases. The chief danger to other children comes from kissing or from using bottles, spoons, or cups which have been infected; as the syphilitic infant is chiefly dangerous on account of the lesions in the mouth. Trouble most frequently

occurs because of ignorance regarding the nature of the disease. It is possible for a syphilitic child to nurse a healthy woman without communicating syphilis, if the child's mouth is treated and the nipple not allowed to become fissured; but it is an experiment which should never be tried.

**Treatment.**—This should always be begun as soon as the first positive symptoms of syphilis appear. Under certain circumstances it may be advisable not to wait for symptoms; as, for example, when both parents have recently suffered from active symptoms, when previous children have died soon after birth, or when, with marked symptoms in the parents, the child exhibits the cachexia of syphilis, but no definite local symptoms. Such anticipatory treatment need not be continued longer than six weeks unless symptoms appear.

The indirect treatment, designed to reach the child through the mother's milk, has fallen into deserved disuse, as it is very uncertain and altogether unsatisfactory.

Mercury is as much a specific for hereditary as for acquired syphilis. There are many ways of introducing it into the system: it may be given by inunctions, by the mouth, by fumigations, by baths, or hypodermically. In most cases inunction is the manner to be preferred in young infants. Gr. x of mercurial ointment, diluted with the same amount of vaseline, may be rubbed daily into the palms, soles, axillæ, or the inner surface of the thighs. It is advisable to change the place of inunction from day to day; and if this is done, it is extremely rare that erythema is produced. If for any reason inunctions are objectionable, as they may be when the family are to be kept in ignorance of the treatment, either the gray powder or the bichloride may be given by the mouth. The usual dose of the gray powder should be gr.  $\frac{1}{2}$  four times a day; that of the bichloride gr.  $\frac{1}{6}$  four times a day, always well diluted. It is rare that larger doses are advisable. When the symptoms are urgent, it is often best to substitute calomel for a few weeks, as the system can usually be brought more rapidly under the influence of mercury by this than by the other preparations mentioned; gr.  $\frac{1}{6}$  four times a day is the usual dose required. Other methods of administration and other preparations offer no advantages, and have some very obvious disadvantages.

The iodide of potassium is to be used, either alone or in combination with mercury, whenever such lesions exist as are classed among adults as tertiary. This includes all the late manifestations, and the earlier ones whenever the bones or viscera are affected. The iodide is usually well borne by children, and may be given in almost any desired dosage. In infancy it is rare that more than twenty grains daily are required, but in older children the necessary amount may be from one to two drachms daily. It should always be given largely diluted.



The duration of mercurial treatment should be at least one year. The doses during the last six months may be reduced to one-half or one-third those employed while active symptoms are present. Treatment should be longer than a year if symptoms exist. It is often better not to give the mercury continuously, but with short periods of intermission.

Ehrlich's salvarsan is quite as efficacious in infants as in older patients. Experience has shown that a single dose does not cure syphilis. A repetition is necessary; two or more injections should be given, and the best results are obtained when it is combined with the mercurial treatment. In older children the intravenous method of administration is to be preferred; an alkaline solution should be employed. For a child of five years the dose is gramme 0.1 or gramme 0.2. In infancy the difficulties in the way of intravenous administration are so great that the remedy must in most cases be injected into the muscles. For this purpose suspension in a bland oil, such as benzoinol, is preferable to solutions, or aqueous suspensions. The best site for injection is the outer part of the buttock high enough to avoid the sciatic nerve. Before removing the needle from the tissues, a few drops of saline solution should be injected through it so as to leave none of the salvarsan in the subcutaneous tissue as the needle is withdrawn; otherwise sloughing may result. The dose for an infant is gramme 0.03 to gramme 0.05.

The tonic treatment of syphilis is important and should not be neglected. After specific treatment has been carried on for a time, particularly if rapidly pushed, the child often becomes anæmic, and suffers greatly from general malnutrition. Under such circumstances it is often wise to discontinue mercury altogether for a time, or at least to reduce the dose very much, and administer cod-liver oil, iron, and other tonics. Such a change is frequently found to act most beneficially, even when lesions are present, which perhaps have been very little or not at all affected by the specific remedies employed. A judicious combination of specific and tonic treatment is required in every case, whether the remedies are given simultaneously or alternately.

*Local Treatment.*—Ulcerative lesions of the skin require cleanliness, dusting with calomel or iodoform, or bathing with the black wash. Mucous patches should be dusted with equal parts of calomel and bismuth. Fissures and ulcers of the mucous membranes should be treated by nitrate of silver. Phagedenic ulcers of the palate or nose should be cauterised with nitric acid or the acid nitrate of mercury. The late syphilitic ulcers of the skin, due to the breaking down of gummata, should be treated aseptically.

## CHAPTER XII.

## INFLUENZA.

*(La grippe.)*

INFLUENZA is an infectious, communicable disease, which is now generally admitted to be due to the bacillus described by Pfeiffer in 1892. It is serious in children chiefly from its tendency to complications of the respiratory tract.

**Etiology.**—The influenza bacillus is found in the secretions of the lower air-passages, less frequently in those of the rhino-pharynx, occasionally in the discharge of acute otitis, rarely in empyema and meningitis. In meningitis the organism is generally found in the blood, i. e., it is a part of a general influenza septicæmia. In the sputum its presence can be demonstrated with certainty only by cultures upon blood agar. In acute cases it may disappear very early; in protracted cases its presence can often be demonstrated for weeks or even months. Besides the bacillus of Pfeiffer, there are usually found in patients suffering from influenza, the pneumococcus, the staphylococcus aureus, and the streptococcus, either separately or in combination. It is often difficult in these mixed infections to tell what part the different organisms play in the pathological process.

Influenza is highly contagious and is almost invariably transmitted by direct contact. In New York the disease attracted little attention until the great epidemic of 1891, since which time it has regularly been seen every winter season with greater or less severity. It disappears with the advent of warm weather. Epidemics prevail chiefly in winter and spring. All ages are liable to the disease, infants under one year especially so.

The period of incubation is uncertain. It is usually short, generally from one to seven days. Little if any immunity seems to be afforded by one attack; recurrences and second attacks are not uncommon in the same epidemic.

**Lesions.**—There are no characteristic lesions of influenza; those which are most frequently found are due to inflammations of the respiratory tract which differ little from the same inflammations when due to other organisms. In some cases the upper respiratory tract is alone or chiefly involved. These cases are frequently complicated by otitis, although the influenza bacillus is not often found in the aural discharge. In other cases only the lower respiratory tract is involved, the process usually spreading in infancy to the lungs, resulting in broncho-pneumonia.

**Symptoms.**—The symptoms of influenza are due to the systemic effects of a general infection, and to certain local inflammations which

may be regarded as complications. The two classes of symptoms—the general and the local ones—are found in all possible combinations.

The milder attacks last from two to five days, occasionally a week. The onset is usually abrupt, with chilliness, muscular pains, and sometimes vomiting. The temperature ranges from 101° to 103° F. Even though the fever is not high, the prostration is considerable, and children are often ill enough to remain in bed for several days. The usual general symptoms which accompany fever are present. Convalescence is frequently protracted, and it may be three or four weeks before the general health is regained. Often there is in addition a mild coryza at the outset and a slight but persistent cough.

More severe attacks are characterised by higher temperature, but only moderate prostration. They often resemble cases of pneumonia,

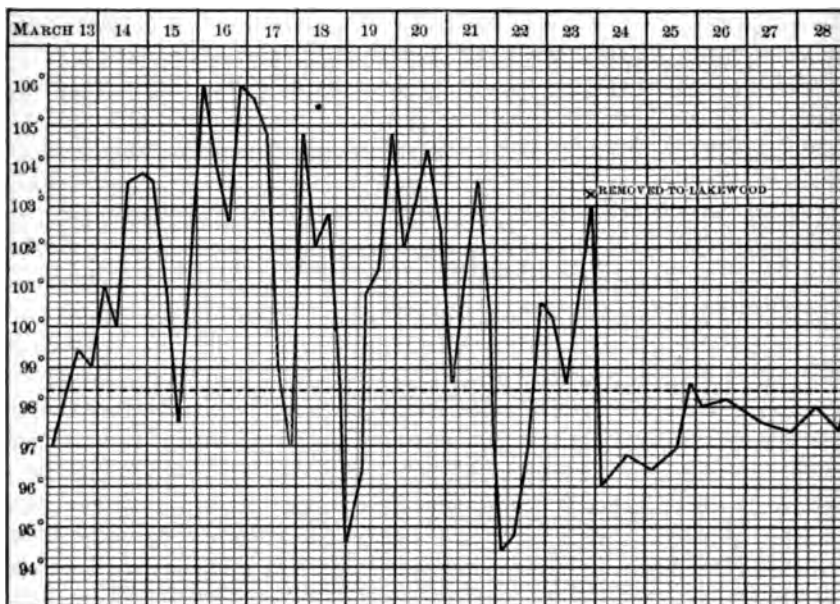


FIG. 216.—TEMPERATURE CHART OF UNCOMPLICATED INFLUENZA. Infant fourteen months old. No local signs of disease; repeated blood examinations for malaria negative; the wide fluctuations of the temperature independent of therapeutic measures. Prompt cessation of fever on removal from the city.

except that the local symptoms and physical signs in the chest are wanting. The onset is usually abrupt with vomiting and headache, rarely with convulsions. The temperature ranges from 100° to 106.5° F. It seldom remains steadily high, but fluctuates widely, often being sub-normal. I have repeatedly seen a temperature of over 106° F. in uncomplicated influenza. Marked nervous symptoms are sometimes present; there may be headache, stupor, and convulsions—symptoms

somewhat suggesting meningitis, but not so continuous as in that disease. More frequently, however, one is struck by the disproportion existing between the high temperature and the general symptoms. The course of the temperature is unlike that seen in any other disease. It is high and fluctuates widely and irregularly without apparent reason. Variations of six or seven degrees in the course of a few hours are very often seen. Often, although the temperature rises every day to 104° or even 105° F., the patient may seem to be scarcely ill at all. The usual duration of these severe attacks is from five to ten days; but even when no complication develops symptoms may last much longer, sometimes until a change of climate is made. (See Fig. 216.) Although the symptoms are very alarming, except in young infants, the attacks are seldom fatal unless pneumonia develops.

Besides these general manifestations the symptoms of acute rhinopharyngitis may be present. The whole pharynx may be the seat of an acute, erythematous blush, or the mucous membrane may present a granular or spongy appearance. Occasionally there is follicular tonsillitis. These catarrhal symptoms may last for several days and gradually subside.

A moderate amount of inflammation of the mucous membrane of the larynx, trachea, and large bronchi occurs in most of the cases of influenza.

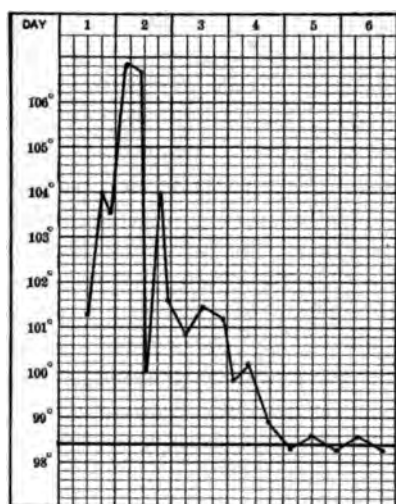


FIG. 217.—ACUTE BRONCHO-PNEUMONIA, ABORTIVE TYPE, COMPLICATING INFLUENZA IN AN INFANT SIX MONTHS OLD. The entire left lung posteriorly was involved.

In the more severe forms, broncho-pneumonia often develops. Sometimes the pulmonary symptoms do not appear for two or three days, or even a week; at other times they are coincident with the development of the fever and other constitutional symptoms, and, except for the prevalence of influenza, this would not be considered a factor in these cases.

The broncho-pneumonia complicating influenza may not differ essentially from the ordinary types, except that the proportion of cases which do not go on to the development of areas of consolidation is larger than is seen under most other conditions. If lobar pneumonia develops, it frequently runs its regular course. But besides these two varieties

of pneumonia, quite a large number of cases of an irregular type are seen with influenza. These are often of short duration, but accompanied by extremely high temperature (Fig. 217).

Vomiting and diarrhoea are frequent at the beginning of influenza, and in some cases, especially in infants, they may continue throughout the attack.

Protracted and recurring attacks of influenza are exceedingly common and the influenza bacillus may be demonstrated for months in the secretions of such patients. The protracted cases in my experience have almost invariably been preceded by a well-defined acute attack, after which there is improvement but not recovery, and an irregular low fever follows, which may drag on indefinitely or there may recur, at intervals of a few days or weeks, periods of very high temperature sometimes accompanied by pulmonary symptoms and signs and sometimes not. The cases are often called malaria, or chronic intestinal poisoning, and not infrequently tuberculosis is suspected; but the special features of all these diseases are wanting. In the cases I have seen the symptoms have been controlled by change of climate, but without this they have not infrequently continued until the following warm season. The rare cases of influenza in which the organisms are found in the blood are characterised by severe constitutional symptoms. They usually prove fatal either from the development of extensive pneumonia or from meningitis. The physical signs in influenza pneumonia and the nervous symptoms in influenza meningitis are not characteristic. Occasionally with severe infections abscesses may develop in the large joints.

**Complications and Sequelæ.**—The most frequent complications are—pneumonia, otitis, and adenitis. Cutaneous eruptions are not infrequent, and are often very puzzling. There may be a general urticaria, or an erythema which sometimes simulates measles, but more frequently scarlet fever. In most of the cases with high temperature the urine contains albumin, and acute nephritis is not infrequent. I have seen three cases of hæmorrhagic nephritis in a single season. All recovered promptly. The nervous sequelæ of adults—mental disturbances, multiple neuritis, etc.—are extremely rare in childhood, although they have been observed. One of the most frequent sequelæ is anæmia; this may be very severe. Following the inflammation of the mucous membranes, there may be chronic enlargement of the cervical lymph glands. Attacks of influenza bear the same relation to the development of tuberculosis as do those of measles.

Convalescence after influenza is usually very slow, and it is often many months before the full effects of a severe attack have disappeared. For a long time the mucous membranes are in an extremely sensitive condition. Relapses are often brought about by slight exposure before the symptoms have quite disappeared, and I have seen them occur from a single outing.

**Diagnosis.**—The ordinary head colds even when severe and epidemic are very rarely due to influenza infection. There are certain features

which distinguish influenza infections of the lower respiratory tract from those due to other causes: these are a tendency to chronicity, to relapses, and to recurrences. In the febrile cases a very high and widely fluctuating temperature accompanied by few constitutional symptoms is always suggestive in the winter season. Recurring attacks of pneumonia separated by an interval of days or weeks with partial or apparently complete recovery are very often due to influenza.

Influenza can be differentiated from the catarrhal inflammations due to other causes only by cultures upon blood agar. These should be made from the bronchial secretion which is obtained as in cases of tuberculosis (q. v.). A culture made from the pharyngeal secretion is not conclusive. Influenza may be confounded with malaria or cerebro-spinal meningitis; from both of these it is distinguished by the methods of diagnosis used

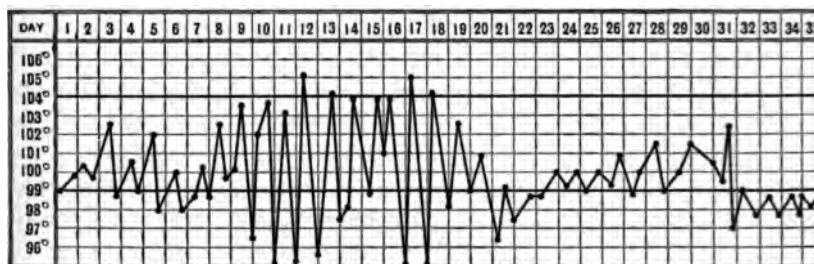


FIG. 218.—INFLUENZA-BRONCHITIS; DOUBLE OTTIS; LATE BRONCHO-PNEUMONIA; AUTOPSY. Infant, nine months old, admitted with influenza-bronchitis; double paracentesis fourth day, repeated on tenth day; the left ear opened again on twelfth and twenty-fourth days. The only signs in the chest were those of bronchitis until the eighteenth day, then broncho-pneumonia which persisted until death. On account of the wide fluctuations in temperature from the eighth to the eighteenth day, mastoiditis and sinus thrombosis suspected. Operation not permitted, partly because of the child's poor condition, but chiefly because the bacillus influenzae was constantly present in the bronchial secretion and this was regarded as a sufficient explanation of the temperature. *Autopsy*.—Moderate broncho-pneumonia; cultures from the lungs showed the influenza bacillus and pneumococcus. Careful examination of the mastoid and sinus showed no trace of disease.

to identify these diseases. Especial difficulties of diagnosis often exist when influenza is complicated by otitis. Although the operation of paracentesis may relieve the local condition it does not arrest the general infection, and the characteristic fluctuations of the temperature belonging to influenza may continue. Under such circumstances the diagnosis of mastoiditis or sinus thrombosis is often erroneously made. (See Fig. 218.)

**Prognosis.**—Uncomplicated cases are seldom fatal, except in infants under six months old; and even though the temperature is very high and the symptoms severe, recovery may be predicted as long as there is no evidence of serious complications. The prognosis of the pneumonia of influenza is rather worse than that of simple broncho-pneumonia. In a

word, influenza is particularly serious in the very young, or when there are pulmonary complications, but rarely otherwise.

**Treatment.**—The communicability of the disease makes it desirable that cases of influenza should be isolated whenever practicable, and particularly that delicate children, or those prone to pulmonary disease, should not be exposed. The fumigation of apartments after attacks should be regularly practised, preferably with formalin gas; this with isolation will do much to control house epidemics.

As there is no specific for influenza, the indications are to sustain the patient, to make him comfortable during the attack, and to prevent so far as possible the occurrence of complications. Every child with influenza should be put to bed and kept there during acute symptoms. At the outset the bowels should be opened by castor oil or calomel. A very high temperature should be relieved by cold sponging or the cold pack, precisely as in pneumonia, but large doses of antipyretic drugs are to be avoided. The nervous symptoms—restlessness, pain, headache, and other disturbances—are best controlled by phenacetine in combination with codeine—e. g., to a child of one year, phenacetine gr. j, codeine gr.  $\frac{1}{16}$ , every three or four hours. Double the dose may be given to a child of two years. Stimulants are required whenever the pulse shows signs of weakness. They should be given according to the same rules as in pneumonia.

The cough which so often persists after influenza is best controlled by cod-liver oil and creosote, used as after acute bronchitis. With persistent bronchitis which resists ordinary remedies, a patient should be sent to a warm, dry climate. The complications of influenza are to be treated as they arise, in the same manner as when they occur under other conditions. In all cases careful feeding in accordance with the general rules laid down for feeding in acute diseases, good nursing, and care to avoid exposure during convalescence, are essentials in treatment. One should be particularly anxious about patients who have a strong tendency to tuberculosis, and such cases should be watched with the greatest care.

In prolonged or constantly recurring attacks nothing is of much avail except a removal to a warm climate. If this is impossible, a young or delicate child should be kept indoors during the cold season, but frequently moved from one apartment to another.

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## CHAPTER XIII.

### MALARIA.

MALARIA is an infectious disease due to the presence in the blood of a specific organism often called the *plasmodium*, but more exactly the

*hæmatocytozoön malariae*. It manifests itself in children by the ordinary acute febrile attacks which are seen in adults and by chronic malarial poisoning. Both of these forms may present certain peculiar symptoms dependent upon the age of the patient.

**Etiology.**—The malarial organism was discovered by Laveran in 1881: it is a parasite of the blood and belongs to the group of protozoa. It is now well established that the parasite enters the blood through the bite of certain forms of mosquito, those belonging to the genus *Anopheles*, and probably in no other way. For this knowledge we are indebted chiefly to the work of Ronald Ross, in India, in 1897. For a general discussion of the malarial parasite, its methods of staining, etc., the reader is referred to works on clinical medicine.

Malaria affects all ages, even the newly-born infant. We must accept with some allowance the statements made by the older writers upon the subject of intra-uterine infection, but in the following case occurring in the practice of my former associate, Dr. Crandall, there seems little doubt that the disease was contracted *in utero*: For ten days before delivery the mother had suffered from a tertian intermittent of moderate severity. Eighteen hours after birth the child was noticed to have cold hands and feet, blue lips and nails, and a pinched face. These symptoms lasted about half an hour and were followed by a distinct fever. Upon the following day the paroxysm was repeated. Examination of the blood of both mother and child revealed the malarial organisms in both cases.

Malaria is more frequently overlooked in young children than in later life, from the fact that its forms are more irregular, and this has led to the belief that young children are less liable than adults to the disease. I believe, however, the opposite to be the case. In a large number of instances where families have been exposed to malarial poisoning I have noted that the young children were frequently the first to show the symptoms of the disease.

Malaria is an endemic disease prevailing in certain localities. Exact knowledge regarding the mode of infection has cleared up many obscure points in its etiology. The rôle of the mosquito explains the greater liability to contract malaria after sunset and during the night, the danger from stagnant ponds and pools of water, the peculiar susceptibility of infants and young children, and the greater frequency of the disease in the spring and summer. Malarial attacks may, however, occur at any season, since the organism may be latent in the body for an indefinite time; how long it is impossible to say, but there seems to be conclusive proof that it may be for many months. Attacks of malaria very often occur when the general health has been reduced by some other cause, particularly by disturbances of digestion.

**Lesions.**—Opportunities for a study of the peculiarities of the lesions of malaria in children are infrequent, especially in New York, as fatal





The hot stage is usually characterised by a higher temperature than in adults, and this is followed by the sweating stage, which is generally marked. The paroxysm may be repeated every day or every other day until controlled by quinine, or the stages may become less and less dis-

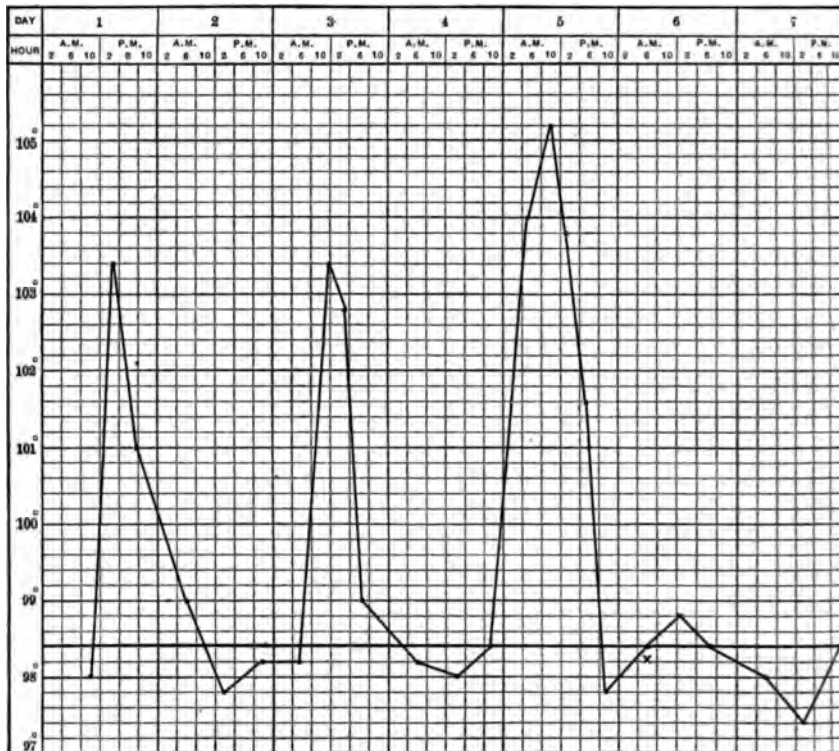


FIG. 220.—TYPICAL MALARIAL TEMPERATURE, TERTIAN TYPE, IN A BOY FIVE YEARS OLD. Onset with vomiting and drowsiness, but no chill. This was an anticipating intermittent, the first paroxysm occurring at 3 P.M., the second at 12 M., the third at 10 A.M.; X marks the time when quinine was begun.

tinct as the disease progresses until a more or less remittent type of fever develops. Less frequently the fever is remittent from the beginning and the constitutional symptoms are of greater severity. In this form there is marked prostration, the tongue is thickly coated, there are often tenderness and pain in the region of the liver, and occasionally there is slight jaundice.

In infants and very young children peculiar types of malaria are seen. A well-marked intermittent fever with distinct stages is often absent, many cases assuming more of a remittent type or an irregular form of intermittent (Fig. 221). The onset is usually abrupt with vomiting, a well-marked chill being rare. Malarial chills are not often wit-

nessed in children under five years old. They are replaced in infants by cold hands and feet, blue lips and nails, sometimes slight general cyanosis, pallor, drowsiness, and prostration. Vomiting has been present in two-thirds of my own cases. Several times I have seen a malarial attack ushered in by convulsions.

The fever is relatively higher than in adults, rising rapidly to  $104^{\circ}$  or  $105^{\circ}$  F., occasionally to  $106^{\circ}$  or  $106.5^{\circ}$  F. This continues from four to twelve hours and gradually falls, usually to normal. The other constitutional symptoms of the febrile stage are much less severe than in most diseases with the same elevation of temperature. The sweating stage is only slightly marked and is often absent altogether. With the fall

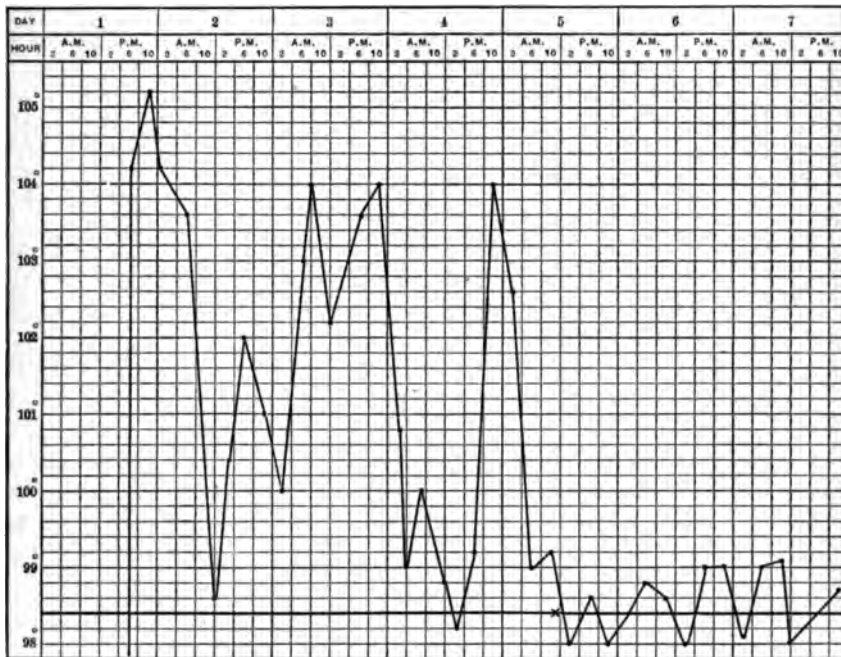


FIG. 221.—AN IRREGULAR MALARIAL TEMPERATURE IN A CHILD NINE MONTHS OLD. The paroxysm on the fourth day was accompanied by an attack of acute pulmonary congestion which came near being fatal; X marks the time when quinine was begun. Although the course of the temperature is irregular, it touched the normal line both on the second and fourth days.

in the temperature there is a gradual subsidence of all the other symptoms of the febrile stage.

After the first paroxysm the patient may be quite well for several hours or even for a day, when the second paroxysm occurs. This is generally not so well marked as the first one, the third may be even less so, and the case may resemble more and more one of continuous fever with wide oscillations in the temperature. In some cases it is remittent at

first and later becomes intermittent, but it is very rare under any circumstances that the temperature does not touch the normal point at some time in the twenty-four hours. In infants the quotidian has been in my experience very much more frequent than any other type, the tertian being uncommon and the quartan almost unknown.

Enlargement of the spleen is present in the great majority of cases, and usually to a sufficient degree to be readily appreciated by examination. The most satisfactory method of examination is by palpation. A spleen which can be easily felt below the ribs (except in the rare cases in which the organ is displaced downward by some condition in the thorax) is enlarged. When it is not sufficiently enlarged to be readily felt by a practised observer under favourable conditions for examination, it is not large enough to be of any diagnostic importance. None of the other symptoms occurring in malarial fever are characteristic; they are quite similar to those which are seen in almost all febrile attacks. They are anorexia, coated tongue, constipation, and restlessness.

**Masked or Irregular Forms of Malaria.**—These are quite frequent in young children, and are due to the presence of certain special or uncommon symptoms which may readily lead to a mistake in diagnosis. They are more often seen than cases of true malarial cachexia.

Among the most frequent of the irregular forms are those relating to the nervous system. Headache is exceedingly common and is usually frontal. When severe and associated with continuous drowsiness, vomiting, and constipation, it may lead to a strong suspicion of tuberculous meningitis. Vertigo is not a frequent symptom, but it is sometimes very prominent. Pains in various parts of the body are very common. A sharp, severe pain at the epigastrium is frequent at the beginning of a paroxysm. It is often associated with tenderness, but has no relation to meals. Less frequently, pain is localised in the region of the spleen or liver. Trifacial neuralgia of malarial origin is rare in childhood. Aching or dragging pains in the muscles of the lower extremities are frequent symptoms during acute attacks, but they are of short duration, disappearing with the fever. They are to be distinguished from the acute lancinating pains of multiple neuritis, which is occasionally seen as a result of malarial poisoning. I have seen the latter in young children in three cases, and it has been observed by others. The pain is accompanied by tenderness of the muscles and nerve trunks, and by loss of power, which is usually partial. Spasmodic torticollis I have seen in eight cases, in which the condition seemed very clearly to depend upon malaria.

Accompanying the paroxysm of malaria there is occasionally seen, more often in infants than in older children, acute pulmonary congestion (Fig. 221), which may give rise to obscure and often very alarming

symptoms. There is an acute onset with vomiting and prostration, high temperature, cough, rapid respiration, and often slight cyanosis. On examination of the chest there is found feeble or rude respiration over one lung, or over both lungs behind, and sometimes coarse moist râles; these signs and symptoms may disappear in the course of a few hours with the fall in temperature, to return with the next paroxysm, or if quinine is given they may disappear entirely.<sup>1</sup> This group of symptoms has sometimes led to the mistaken opinion that the disease was pneumonia, which had been aborted by the administration of quinine.

**Subacute or Chronic Forms of Malaria.**—The most constant symptoms are anæmia, enlargement of the spleen, and slight fever. The anæmia is usually marked, often being extreme. The enlargement of the spleen is distinct, easily made out by palpation, and sometimes is very great. The fever is often so slight as to be discovered only when the temperature is taken five or six times in the twenty-four hours. The other symptoms are of a very indefinite character; there may be slight œdema of the lower extremities, general muscular weakness, so that the child is easily fatigued, loss of appetite, coated tongue, constipation, headache, muscular pains, and often cough from a slight bronchitis. These symptoms may depend upon many conditions other than malaria, even when they are seen in a malarial district. The only positive evidence of malaria in such cases is the presence of the malarial organisms in the blood. Even the swollen spleen, anæmia, and slight fever, which are often looked upon as diagnostic, may be present in cases of anæmia with which malaria has nothing whatever to do.

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<sup>1</sup> The following case is a good example of this condition in its more severe form, and illustrates the difficulties in the diagnosis of malaria in infancy: A fairly nourished child, nine months old, who had been under observation in an institution for two weeks, was suddenly taken with vomiting and fever (Fig. 221). A cathartic was followed by a large undigested stool, and as the temperature then fell to normal, the attack was regarded as one of indigestion. On the third day the temperature was again high and accompanied by cough; coarse râles were found throughout the chest, and fine râles at the right base; it was then thought that pneumonia was developing. On the fourth day all the symptoms were so much improved that the infant was regarded as convalescent. At 6 p. m. the temperature was normal, and the infant went to sleep quietly. At 9.30 p. m. he awoke with a temperature of 104° F., extreme restlessness, and marked dyspnoea. In half an hour his symptoms had increased to a point where he seemed likely to die. He became cyanotic, the respirations were of a panting character and rose nearly to one hundred a minute, and he coughed with almost every breath; the pulse was scarcely perceptible. The severe symptoms continued for about an hour, then passed away gradually, and at the end of two and a half hours they had completely disappeared, and the child was in a quiet sleep which continued until morning. Malaria was now suspected, and the diagnosis established by the discovery of the plasmodium in the blood. The spleen was at this time much enlarged; the signs in the chest were those only of bronchitis of the large tubes. Quinine was given in full doses, and immediately controlled the temperature and the pulmonary symptoms.

**Diagnosis.**—The positive diagnosis of malaria rests upon the demonstration of the malarial organisms in the blood. They will be found in nearly all the cases provided a careful examination is made a few hours before the paroxysm, and also that no quinine has been administered. When their number is small they may be missed at the height of the fever, although they may readily be found just before the temperature begins to rise. While a positive result is conclusive, a negative one is not always so because of the impossibility of fulfilling all the above conditions. This fact and lack of experience in blood examinations make it necessary for a large part of the profession to make the diagnosis by the other symptoms. These, in the order of their importance, I would place as follows: Prompt curability (especially in cases of fever) by quinine; distinct periodicity in the symptoms; enlargement of the spleen; and a history of an exposure in a district known to be malarial. Particular importance is to be attached to the therapeutic test. Recent experience emphasises more and more strongly the fact that quinine has very little influence upon fevers which are not malarial, and, conversely, that a fever immediately and permanently controlled by quinine is pretty certain to be malarial.

The cachexia and course of the temperature in septicæmia, pyæmia, broncho-pneumonia, tuberculosis, and empyema, may cause them to be mistaken for malaria. The fever and recurring chills of pyelitis are often attributed to malaria; as are also the heaviness, lethargy, headache, coated tongue, and slight fever of chronic intestinal indigestion. Many conditions accompanied by an enlarged spleen may be confounded with malaria, especially simple anæmia, leukæmia, rickets, and syphilis. While malaria may be multiform in its manifestations, the physician can fall into no more serious error than to regard all ailments with obscure or indefinite symptoms as malarial, neglecting careful physical and blood examinations, by which means alone an accurate diagnosis is reached.

**Prognosis.**—Although it is seldom fatal in itself, an attack of malaria in a young child may so undermine his constitution that he may succumb to some other acute disease. Cases are often difficult to cure while the patient remains in the malarial districts, and when frequent reinfection occurs. Under other circumstances and with proper treatment the prognosis of malaria is good.

**Treatment.**—*Prophylaxis.*—More exact knowledge regarding the etiology of malaria makes it possible for much to be done in the way of prevention. Besides the general measures proposed for the extermination of the mosquitoes concerned, emphasis should be laid upon the necessity, in the case of young children, of protecting them against the bites of mosquitoes in localities which are or which may possibly be malarial. This can be done by a more thorough use of mosquito netting and by using upon exposed parts of the body lotions or ointments containing

menthol, pennyroyal, turpentine, or other substances which keep these pests away. The general treatment is symptomatic, and is to be conducted as in all acute febrile diseases. In the cold stage, stimulants or a hot bath may be required; in the hot stage, ice to the head and frequent sponging. The bowels in all cases should be freely opened, preferably by calomel.

*Methods of Administration of Quinine.*—For infants my own preference is to give the bisulphate in an aqueous solution, one or two grains to the teaspoonful, according to the age of the patient. Most infants take such a solution with less difficulty and vomit it less frequently than the combinations with the various vehicles supposed to cover its taste. In the event of failure by this method, the same solution may be given *per rectum* through a catheter. It should then be more largely diluted with some bland fluid such as gruel, and in double the dose. This is necessary, not only because absorption is less certain and complete, but also because a rectal dose can seldom be repeated oftener than every five or six hours. There is sometimes an advantage in giving part of the quinine by the mouth and part of it by the rectum; should both fail it may be given hypodermically. For this purpose the bimuriate of quinine and urea, the hydrobromate, or the bisulphate may be used. All are more or less irritating and there usually follows some induration at the site of the injection, which may last a long time. While the hypodermic use of quinine is sometimes invaluable it should not be employed in infants except in serious attacks and when the diagnosis has been established. In a number of instances both in hospitals and private practice I have seen sloughing follow the use of nearly all the preparations generally employed. The occurrence of abscess points to infection at the time of injection; but necrosis I believe may be due simply to the irritation of the quinine upon tissues having a lowered vitality, as in the case of young or delicate infants. I have seen this happen when the strictest precautions against infection were observed. The frequent repetition of the hypodermic injections should be avoided; in most cases, one or two good doses are sufficient, the effect being continued by quinine given by other methods.

For children from two to seven years old the taste of quinine must be concealed. An aqueous solution of the bisulphate may be mixed with the syrup of sarsaparilla, orange, or yerba santa; or the sulphate may be given in suspension in one of the same vehicles, the mixture being made just before the dose is taken; otherwise the partial solution of the drug will render the whole dose exceedingly bitter. When the dose required is not large, as in the milder cases, the lozenges of the tannate of quinine combined with chocolate answer the purpose admirably, for these are so nearly tasteless that children will take them without difficulty. Each lozenge usually contains one grain of the tannate, which is equivalent to

about one-third of a grain of the sulphate of quinine. A similar lozenge containing one grain of the sulphate may be made, which is often taken by children without the slightest objection. The bisulphate may be given in solution by the rectum, or, better, at this age, in the form of suppositories; but, as in infancy, with very urgent symptoms, it is better to resort at once to the hypodermic method in case of failure by the stomach.

For children over seven years old, the same methods of administration may usually be employed as in adults. It is always preferable to give quinine in solution, or if not so, in capsule, but not in pill form.

In a case with well-marked paroxysms the quinine should if possible be given in the interval, with the largest dose about four hours before the expected paroxysm. With infants this plan is sometimes impracticable, as frequent small doses are usually better borne by the stomach than a few large ones. In them also vomiting seems less likely to occur when it is given on an empty stomach. For this reason it is advantageous to give the drug at regular two- or three-hour intervals during the night, and omit all medication during the day.

*Dosage.*—Relatively much larger doses of quinine are required for young children than for adults. Except for its tendency to disturb the stomach, quinine is borne remarkably well by little patients. Generally too small doses are given. An infant of a year with a sharp attack of malarial fever will usually require from eight to twelve grains of the sulphate (ten to fourteen grains of the bisulphate) daily. Occasionally I have found it necessary to give double the quantity referred to, and I have seen no unpleasant cerebral symptoms. It is useless to expect to control an acute attack of malaria by such doses as one grain three or four times a day. Children from five to ten years old require almost as large doses as do adults. None of the substitutes for quinine are to be relied upon in acute cases.

In chronic cases, arsenic and iron are usually required in combination with smaller doses of the quinine than those mentioned. For children over seven years old, Warburg's tincture may be employed. In most chronic cases a cure can be effected only by a change of climate.

The masked and irregular manifestations of malaria are to be treated in the same manner as cases of malarial fever.



## SECTION X.

### OTHER GENERAL DISEASES.

#### CHAPTER I.

##### *RHEUMATISM.*

THE rheumatic diathesis manifests itself in children by quite a different group of symptoms from those seen in adults; for this reason the disease was formerly supposed to be a rare one in early life. It is only within recent years that its frequency and its peculiarities have come to be appreciated. For our present understanding of the subject we are indebted largely to the work of English physicians, especially Cheadle, who has brought out more fully than any one else the close connection existing between many conditions formerly not regarded as rheumatic. One who has in mind only the adult types of articular rheumatism, and regards arthritis as a necessary symptom for a diagnosis, will overlook in early life many manifestations which are clearly the result of the rheumatic poison. There is seen at this period a group of clinical phenomena, which often occur in combination or in succession, whose association was not understood until they were all discovered to be related to rheumatism. Sometimes one member of the group and sometimes another is first seen, but when one has appeared others are likely soon to follow.

Rheumatism in childhood, then, is manifested not alone by arthritis with acute or subacute symptoms, but by a large number of other conditions which are not to be regarded in the light of complications, but rather as forms of the disease.

**Etiology.**—It is not in the province of this work to discuss the various theories regarding the nature of rheumatism and its exciting cause. The drift of medical opinion to-day is strongly toward the view that acute rheumatism is an infectious disease, probably of microbic origin. Although the character of the micro-organism is not yet determined, the latest observations of Poynton and Paine<sup>1</sup> point to a diplococcus. The excessive formation of acids in the system may be regarded as a result of the infection, or possibly as a condition necessary for the activity of

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<sup>1</sup> *Lancet*, May 4, 1901.

the specific poison. Under five years of age articular rheumatism is not common, and in infancy it is extremely rare. I once saw, however, in a nursing infant, a typical attack of rheumatic fever with multiple joint lesions. The condition is, however, so exceptional that one should be cautious in making the diagnosis of rheumatism in infancy. Most of the cases so regarded are examples of scurvy. After the fifth year both the articular and the other manifestations of rheumatism become very common, and occur with increasing frequency up to the time of puberty.

Heredity is a very important etiological factor, and in fully two-thirds of the cases that have come under my care, a rheumatic family history was obtained. Of the other important causes, the most frequent are living in damp dwellings, direct exposure to cold and wet, poor hygienic surroundings, and insufficient food. While seen among all classes, rheumatism is more common among those who are badly housed. Attacks of rheumatism occur at all seasons, but are much more frequent in the spring months. One attack strongly predisposes to a second, and in most cases there is a history of a large number of attacks of greater or less severity. Among my own patients, girls have been affected with greater frequency than boys.

**Symptoms.**—*The General and Articular Manifestations.*—The clinical types of rheumatism in children present very notable contrasts to those seen in adults. A typical attack of acute articular rheumatism such as is seen in adult life, with a sudden onset, high temperature, severe inflammation of several joints, profuse acid perspiration, and occasional delirium, is rarely seen in a child under eight or ten years old. In most of the attacks in childhood the onset is not very acute, the temperature is but slightly elevated—only 100° or 101.5° F.—the swelling and pain are moderate, and the redness is often absent. The number of joints involved is generally small, those most frequently affected being the ankles, the knees, the small joints of the foot, the wrists, or the elbows. These symptoms are often not severe enough to keep the patient in bed, and only the pain in the joints of the lower extremities prevents him from walking. The duration of these attacks is from one to three weeks, and in the course of a month most of them recover even without treatment.

Not infrequently the symptoms are limited to a single joint, usually the hip, knee, or ankle. Possibly the joints of the upper extremity are affected oftener than would appear, but disease here is much more likely to be overlooked than when lameness is present. The swelling is moderate and may not be evident except on a close examination; in some cases there is none. There is stiffness of the joint, as shown by lameness, and there may be so much pain and soreness that the child refuses to walk altogether. Muscular spasm about the affected joint is often marked, and may be the most striking objective symptom. The tenderness is sometimes localised, but it may affect the ligaments, tendons, and

even the muscles. These symptoms may persist for two or three weeks and lead to a suspicion of incipient tuberculous disease of the joint. Rheumatism is distinguished by its more acute onset and usually by the presence of slight fever; some elevation of temperature being the rule, though it is not often much over 100° F. A family history of rheumatism, or a history of previous similar attacks in the patient affecting the same or other joints, or other manifestations of rheumatism, are also of assistance in the diagnosis. Occasionally all doubt is removed by the disease extending to other joints, or by the development of endocarditis. In some cases the symptoms are less in the articulation than in the muscles, and they are dismissed as simply "growing pains," having nothing characteristic about them except their occurrence in damp weather.

*Cardiac Manifestations.*—These may occur when the articular symptoms are very mild, and in some cases when they are entirely absent. The most frequent is endocarditis. This is much more often seen in the acute rheumatism of children than of adults, and probably occurs in the majority of all severe cases; if it does not come in the first attack, it is likely to be seen in the later ones. It frequently occurs with a mild rheumatic arthritis, often being unnoticed until valvular disease of considerable severity has developed. Sometimes there is only high fever with severe constitutional symptoms of an indefinite character, but no arthritis, and no suspicion that the attack is rheumatic until endocarditis is discovered. Such cases are not infrequent. If the patients are kept under observation, articular symptoms are almost certain to develop later, and often there are other manifestations of rheumatism, especially chorea.

Pericarditis is much less frequent than endocarditis, and usually occurs in children over seven years old. It is often associated with endocarditis. The most characteristic form of inflammation in early life is a sub-acute, dry, fibrous form, often resulting in great thickening with extensive adhesions, and frequently in obliteration of the pericardial sac. When once started it shows a strong tendency to recurrence and persistence.

The heart is so frequently affected in the rheumatism of childhood that it should be closely watched whenever articular symptoms are present, no matter how mild they may be; and not only in these cases, but in all the conditions hereafter enumerated with which rheumatism is likely to be associated.

*Inflammations of other serous membranes*—the pleura, peritonæum, and pia mater—were much more frequently ascribed to rheumatism in the past than now. There is little doubt that on rare occasions any one of these may be due to rheumatism. The pleura is most often involved, but even this is rare in young children.

*Torticollis* when it occurs acutely is frequently rheumatic. This form is characterised by its sudden development, continuous spasm, the

great amount of muscular soreness, the moderate pain, and the fact that it usually disappears spontaneously after a few days. Other manifestations of muscular rheumatism are less characteristic and usually affect the muscles of the extremities.

*Anæmia* is almost invariably seen in rheumatic patients, both during and between the attacks. The effect of the rheumatic poison upon the blood resembles that of malaria. The presence of anæmia is so evident and its degree often so marked, that one may have great difficulty in distinguishing cardiac murmurs which are hæmic from those due to endocarditis.

*Chorea*.—In the article upon Chorea I have already discussed the association of that disease with rheumatism and expressed my own belief in a very close relationship existing between them. Not very infrequently chorea is the first manifestation of a rheumatic diathesis, to be followed soon by articular symptoms or by endocarditis without such symptoms. In other cases chorea and acute endocarditis occur together without articular symptoms, or all three may be associated. Whichever of the three conditions is first seen, the physician should always be on the lookout for the others. The frequency of rheumatism in choreic patients has been variously estimated by different observers; in my own cases over fifty-six per cent gave unmistakable evidence of a rheumatic diathesis.

*Tonsillitis*.—The association of tonsillitis and pharyngitis with rheumatism appears in many cases to be a close one. Children who are the subjects of frequent attacks should be regarded as possibly rheumatic, and closely watched for other signs of that disease. Acute tonsillitis often ushers in an attack of rheumatic arthritis, and occasionally acute endocarditis without articular symptoms. The nature of the relationship is not yet fully explained; by many the tonsils are regarded as the structures through which the rheumatic poison is absorbed.

*Subcutaneous Tendinous Nodules*.—General attention was first drawn to these as a manifestation of rheumatism by Barlow and Warner, in 1881, who described them as "oval, semi-transparent, fibrous bodies like boiled sago grains." They are most frequently found at the back of the elbow, over the malleoli, at the margin of the patella; occasionally on the extensor tendons of the hands, fingers, or toes, or over the spinous processes of the vertebræ or the scapulæ. They are composed of fibrin, cells, and fibrous tissue, and vary in size from a large pin's head to a small bean, sometimes being as large as an almond. The nodules may come in crops, lasting for a few weeks and then disappearing, or they may last for months. An eruption of nodules is usually coincident with other rheumatic manifestations. These nodules are better felt than seen, although they may be visible if the skin is tightly drawn. They are certainly not common in this country; and although I have made it a

rule to examine rheumatic patients for them, I have seen them but seldom, and they have been prominent in only two or three cases. This, I think, has also been the experience of most observers in New York. From published reports, however, they appear to be much more frequent in England. There can be no doubt regarding the connection of these nodules with rheumatism.

*Erythema.*—The connection between rheumatism and the various forms of erythema—marginatum, papulatum, and nodosum—has been very clearly shown by Cheadle. None of these are frequent conditions in childhood, but when seen they should always suggest rheumatism.

*Purpura.*—The association of purpura with rheumatism is so frequent that there can be little doubt of the close connection between the two conditions. Rheumatic purpura, however, is quite distinct from the other forms of purpura, and is a much less frequent disease.

*Diagnosis.*—In order to recognise rheumatism in a child, one must free his mind from preconceived notions of the disease drawn from its manifestations in adults, as very few cases correspond to the adult type of acute rheumatism. In early life the disease is recognised not by any one or two special symptoms, but by the association or combination of a number of conditions which may appear unrelated. In determining whether or not any given set of symptoms is due to rheumatism, one should consider: (1) The family history, since in early life heredity is so important an etiological factor; (2) the previous history of the patient, not only as regards articular pains and swelling, the slight joint-stiffness without swelling, the indefinite wandering pains in damp weather, and the so-called growing pains, but also the previous existence of chorea, frequent attacks of tonsillitis, torticollis, or erythema; (3) the examination of the patient, which should include a careful search for tendinous nodules, as well as a thorough examination of the heart for signs of endocarditis or pericarditis, and, in cases which are at all acute, the temperature. In doubtful cases with monarticular symptoms much importance is to be attached to the presence of slight fever, the abrupt onset, and tenderness of the neighbouring muscles and tendons—all occurring without a history of traumatism. Rheumatism is more often overlooked than confounded with other diseases; although in childhood multiple neuritis and tuberculous and syphilitic bone disease are often mistaken for it, and in infancy the same is true of scurvy. The extreme infrequency of rheumatism during the first two years of life should always make one sceptical regarding it. In an infant, when the symptoms are confined to the legs and are not accompanied by fever, they are almost certain to be due to scurvy, even though the gums are normal and ecchymoses have not yet appeared. Multiple gonococcus arthritis has often been diagnosed rheumatism.

**Prognosis.**—Rheumatism in a child is in itself seldom if ever dangerous to life. In the great majority of cases the articular symptoms soon disappear, even without special treatment. The danger from the disease consists in its cardiac complications. One attack of rheumatism is almost certain to be followed by others, and when once the heart has been affected its lesions are likely to increase with each recurrence of the disease.

**Treatment.**—Rheumatism in children derives its chief importance from its relation to cardiac disease. Cardiac complications are so frequent and so serious that everything possible should be done to avert rheumatism from those who by inheritance are especially predisposed to it, to prevent its recurrence in a child who has once had the disease, and during an attack to prevent the heart from becoming involved. The relation of diet to rheumatism is very imperfectly understood. The best opinion at the present time is that there is no very close connection between the two. The underclothing should be of flannel during the entire year, in summer the lightest weight being worn. The feet should be carefully protected, and exposure in damp weather avoided. Indoor occupations should be chosen for rheumatic boys.

The tendency to recurrence is so strong in this disease that a child of rheumatic antecedents, who has shown in the various ways mentioned a marked predisposition to rheumatism, and who has had an attack, even though a mild one, should, if possible, spend the winter and spring in some warm, dry climate, or even remain there permanently. Otherwise in most such children, it is only a question of time when, with the repeated attacks, the heart will become involved.

To avert the danger of cardiac complications during an attack of rheumatism, or to limit their extent, there are two things which should invariably be insisted on: First, to confine to the house and in a warm room every child with rheumatic pains, no matter how mild; secondly, if fever is also present, to keep the child in bed while it continues, even though it may never be above 100° F. Absolute rest and the equable temperature thus secured are unquestionably of more importance than anything else in protecting the heart during a rheumatic attack. With these precautions must be combined an early diagnosis. In very many, perhaps in most cases, the harm is done before the true nature of the disease is suspected, the symptoms being dismissed as of slight importance because the articular manifestations are not very severe. Children who have once had rheumatism should be closely watched during chorea and other diseases related to rheumatism, the heart should be frequently examined, and the physician should be on the alert for the first articular symptoms.

Aside from the measures just mentioned, the treatment of rheumatism in childhood is to be conducted very much like that of adult life.

In the most acute attacks either salicylate of soda (gr. v every three hours to a child of five years), oil of wintergreen, aspirin, or salicin should be given; as the majority of cases are not very acute, marked improvement is by no means always obtained by these drugs. Alkalies should be given in all cases in combination with the specific remedies, but particularly in those in which there is hyperacidity of the urine. Either the acetate or citrate of potassium or the bicarbonate of sodium may be used, a sufficient quantity being administered to render the urine alkaline.

Quite as important as these drugs is the use of general tonics, particularly iron and cod-liver oil. These should be given not only between attacks to fortify patients against their recurrence, but also in subacute cases which are sometimes influenced very little or not at all either by salicylates or alkalies.

## CHAPTER II.

### *DIABETES MELLITUS.*

In this chapter will be attempted only a description of the peculiar features which diabetes presents when affecting young patients. It is a very infrequent disease in children. Of 1,360 cases of diabetes collected by Pavy, only eight were under ten years of age. In a series of 700 cases collected by Prout, only one case was under ten years. In a series of 380 cases collected by Meyer, only one case was under ten years of age.

**Etiology.**—Stern, in a series of 117 collected cases of diabetes in children, states that 47 were females and 31 males, the sex in the other cases not being given. Although extremely rare, cases have been observed during the first two years, and even during the first year of life. Statistics on this point are not altogether trustworthy, since some cases of temporary glycosuria have certainly been included.

Among the etiological factors, heredity is one of the most important. Pavy reports the case of a child dying of diabetes at two years in whose family the disease had existed for three generations. Instances have been recorded of the occurrence of diabetes in four or five children of the same family. Inherited gout, insanity, and nervous diseases generally, may be looked upon as factors in the production of diabetes. Several of the cases reported in children have been preceded by injuries received upon the head. In a number of my own cases the disease has followed the consumption of large quantities of sugar for a long time. Often no adequate cause can be found.

**Symptoms.**—The most important early symptoms are thirst, polyuria, and wasting; their development is often quite rapid. The thirst is intense, often leading children to drink four or five pints of fluid a day.

The amount of urine passed varies from one to eight quarts daily. The specific gravity is from 1.026 to 1.040, and the usual amount of sugar is from three to five per cent, rarely more. Albumin is not infrequently present. Incontinence of urine is an important symptom, and often one of the earliest to be noticed. The wasting is usually quite rapid, so that a child may lose as much as six or eight pounds in a month. It is generally accompanied by anæmia. The appetite may be poor; at times, however, it is voracious. Other symptoms of less importance are a dry mouth, scanty perspiration, irregular sleep, occasional epistaxis, furuncles and abscesses, decayed teeth, and genital irritation.

The course of the disease is much more rapid in children than in adults, and, as a rule, the younger the child the more rapid its progress. The majority of the cases prove fatal in from two to four months from the time the symptoms are sufficiently marked to make the diagnosis possible. Very few last more than six months; occasionally, however, one of the milder type may be prolonged from one to two years.

The progress of the disease is marked by continuous wasting, which may result in a marked degree of marasmus, and prove fatal. Some are carried off by intercurrent pneumonia or tuberculosis, but the majority die comatose. When coma develops, the case may be considered hopeless, and death is likely to be postponed but a few days. The cause of diabetic coma has not yet been satisfactorily explained, but it is usually believed to be due to the presence of the acetone bodies in the blood.

**Diagnosis.**—Diabetes is apt to be overlooked, because of the common neglect of urinary examinations in children. The prominent symptoms—thirst, polyuria, and wasting—when associated, should always attract attention. Incontinence of urine, accompanied by marked wasting, is always suspicious. In some cases genital irritation may be the most prominent early symptom. A positive diagnosis is made only by an examination of the urine.

**Prognosis.**—In few diseases is the prognosis so bad as in diabetes in children. So high an authority as Senator declares that diabetes in children is hopeless and all treatment is useless. From a study of seventy-seven cases, Stern reaches the same conclusion. There are, however, cases on record in which recovery is believed to have taken place. The cases which I have seen have all terminated unfavourably. In a given case the prognosis, as to the duration of the disease, is rendered much worse by the presence in the urine of large amounts of diacetic and  $\beta$ -oxybutyric acids. This condition is even more serious than is a high percentage of sugar; that the patient will then live more than three months is highly improbable.

**Treatment.**—The indications for treatment are the same in children as in adults: first, diet; secondly, general hygienic measures. From the use of drugs nothing can be expected.



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